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RETROBULBAR OPTIC NEURITIS.

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(A Clinical Lecture given at the Hospital on March 8, 1899.)

In the cases I propose to speak about the mischief is limited to one optic nerve;* the lesion affects the nerve behind the eye but in front of the brain, and it does not depend either upon changes in the eye itself or upon disease within the skull. The entire class, not a large one, is by no means homogeneous; the cases indeed form a rather mixed assemblage, and one of their most obvious common characters is that they cannot be classed anywhere else. The cases of symmetrical toxic amblyopia—with tobacco amblyopia as the type—are outside our subject.

The optic nerve, like other nerves, is subject to inflammation beginning in itself; whilst, on the other hand, disease may be communicated to the nerve by the various parts amongst which it passes between the chiasma and the eye—the orbital tissues, the optic canal, the sphenoidal sinus. It is therefore likely on pathological grounds that two chief families of the retrobulbar neuritis we are now considering exist; and we find in fact that an assignment of the cases to one or the other of the two pathological families just mentioned is usually justified on clinical grounds.

* Or attacks the other only after an interval.

We must, however, neither think nor speak dogmatically on this subject, for the opportunity of verifying the nature of any case of single retrobulbar neuritis must always be excessively rare—if indeed it should ever occur. And however sure we may feel that the pathological division into an *idiopathic* and a *symptomatic family* is correct, we shall continue to meet with some cases which, though undoubtedly belonging to one family or the other, cannot on clinical grounds, with our present knowledge, be confidently placed in either.

In the retrobulbar neuritis I am speaking of, usually, as already stated, only one optic nerve suffers, and if both should be attacked there is an interval commonly of months, sometimes of years, between the two.

In Case 134* the interval between the first eye and the second was three months; in Case 115, six months; Case 132, about nine months; Case 93, 12 months; Case 116, five years; Case 28, several years. An interval of days only is very infrequent (Case 74).

The failure of sight is generally rapid, the climax being generally reached in from one day to four or five; but it is never really "sudden" as in embolism or arterial thrombosis. The degree of failure of vision varies extremely in the idiopathic cases, from the faintest "mistiness" of objects in the centre of the field to total loss of perception; it bears no uniform relation to the early ophthalmoscopical changes; and recovery of good sight is possible even in the worst cases, provided that the improvement sets in tolerably soon. In the symptomatic family slight forms are much less common and the prospect of recovery not nearly so good.

* The numbers refer to 148 cases, being all of which I have notes, consisting of 123 which were arranged for the discussion on Retrobulbar Neuritis at the Ophthalmological Society (Trans., vol. xvii, 1897), and 25 that have occurred since. Some of the earlier ones were published in a paper in the Transactions of the same Society in 1884 (vol. iv). Of the whole series (148) 28 are omitted as incomplete, or doubtful, and the lecture is therefore based on 120 cases.

The field of vision is invaded in various degrees, but in cases of every degree and kind the central part is involved; and in the slighter varieties this is as a rule the only part affected. The defect is often more or less fan-shaped, spreading towards the periphery though not constantly in the same direction.

Something can often be learnt, particularly in the milder cases, from the patient's description of the defect of sight.

A lady of 34 (Case 63) could see 6/9 with the affected eye, but the letters were "misty" and there was "a dark mark near the centre." Another lady of 35 (Case 66) said that the defect had come on during one day in the form of a "spot" and of "a darkening" of the objects she looked at; when I saw her two weeks later V. was 6/6, but there was still "a patch of mist," and I found a relative scotoma (for red) a little up and in from the centre; a month later colours and light looked alike to the two eyes.

A lady of 29 (Case 68) said that when the sight of her right eye failed a white surface "looked like mottled soap," and that print and coloured objects had the appearance of being "gapped" (interrupted). When seen 10 days after the failure, V. of the affected (R.) eye was 6/18, with much loss of temporal part of field and no ophthalmoscopical changes; pupil not noted; in six weeks V. was 6/9; in five months 6/9, still with no definite changes at O. D.

The direct reflex action of the pupil is lowered, even in the milder cases, more than the loss of visual acuteness seems to warrant, and in the bad cases it is of course almost or entirely lost.

Pain about the affected eye is a very common symptom, though, as in so many other maladies, its value in diagnosis requires adjustment for each patient. It is of two sorts: spontaneous pain behind the eye and in the corresponding temporal district; and pain caused or increased by movements of the eye—sometimes by movement in one particular direction—or produced by pressing the eyeball back into the orbit. Observe carefully that the pain, however bad, is

limited to the side of the affected optic nerve, and is not accompanied by vomiting. In the idiopathic cases the pain usually precedes the loss of sight and ceases when, or soon after, this occurs; it is a less frequent feature in the symptomatic cases, but when present in them is often more severe and much more prolonged, continuing for days or even weeks after the sight has failed.

I have never seen and very seldom heard of any external signs of inflammation, and such slight symptoms of it as are occasionally mentioned by the patient may perhaps be accounted for by his having kept the eye covered or closed during the painful stage.

A young man (Case 26) spoke of the eye having been red during the stage of pain that preceded the failure of sight. Another patient (Case 27) said that the lids had been rather swollen; and a third (Case 52), also a young man, spoke of the eye having been a little blood-shot during the severe pain of the early period.

Some ophthalmoscopical changes* are usually present almost from the first in the idiopathic cases, but they are often very slight and occasionally quite wanting; such early changes—various degrees of haziness of the disc, sometimes with decided swelling, but without hæmorrhages and with only moderate venous engorgement—point to a true, if not as a rule very gross, acute inflammation of the nerve. In some of the worst symptomatic cases, however, no change whatever can be seen at the disc for some weeks after the loss of sight.

Later on in cases of both families the disc almost always becomes pale, often very pale, whether the sight improves or not. In very few is it true that the nerve passes through an

* P.S.—I see that Dr. Sym, in a recent paper on retrobulbar neuritis (American Journ. Med. Sci., February, 1899), states (p. 154) that ophthalmoscopical changes are "usually absent in the early stages and may be absent altogether." I think that a careful comparison of the two discs will show slight but decided differences within a few days of the failure of sight in a large proportion of cases.

attack causing, even for a time, severe loss of sight, without some appearances either of early inflammation or late atrophy of the disc.

The inconstancy in the degree of visible papillitis and in the date and degree of the consecutive pallor doubtless indicate differences not only in the severity of the morbid process, but also in the distance at which it is seated from the eyeball.

In Case 9 (F., æt. 36) the disc was becoming pale two weeks after the onset.

In Case 45 (F., 38) slight haze and swelling of O. D. with V. 6/12 partly, one week after onset: O. D. appeared quite normal and V. 6/6 four weeks after onset.

Case 72 (M., 17) showed congestion and slight haze with V. = shadows at ninth day. O. D. pale and V. 6/12 at end of fourth week.

Case 72 (F., 27) four days after onset, papillitis with swelling = 3 D. and V. = fingers at 3 feet; seven weeks, O. D. pale all over, V. 6/6.

In Case 78 (F., 28), seventh day, haze, but no swelling; four weeks decided pallor.

Case 97 (F., 30) three days slight haze and swelling, V. = fingers eccentrically; four weeks clearer and getting pale V. 6/12.

In Case 82 (F., 29) one week, O. D. slight hazy, V. 6/18, with a large scotoma from centre up and out; six weeks O. D. pale all over and clear.

Cases 68 and 122 showed no decided changes from beginning to end; the former has been already quoted, the latter (122) was as follows:—

Rapid severe failure of one eye with very slight changes at the time or afterwards. Complete recovery in a month. No personal complications, but family history of nervous disease, and optic nerve disease in a brother. Condition five years later.

Mrs. G., æt. 38 (Case 122), was sent by Dr. Hague on January 3, 1884, for recent blindness of the L. eye; she could see my hand in the temporal part of the F., but nowhere else, pupil motionless

to direct light. The only changes were slight haze of O. D., and a decided whiteness of the coats of several of the arteries on and a little beyond the O. D.; all the arteries pulsated easily on pressure (*i.e.*, no indications of arterial blocking); colour of O. D. normal.

She said that as she was at needlework about half past 11 A.M., on December 28 (five days), "a feeling came as if there were something across the sight of the left eye," by 5 P.M. the same day she could not see the V, VI, and VII on the clock face, and by 7 P.M. she "could not see the clock face at all, though she could still see the fire."

Dr. Hague reported that there were no signs of renal or other organic disease. She had often had "rheumatism" in knees, wrists, and shoulders since she was 15, but never rheumatic fever; had always been subject to bad megrim attacks, consisting of headache on the L. side with "forcing" pain in the eye, followed by pain at back of neck and profuse micturition, sometimes with nausea, but no vomiting; the visual spectra of megrim had never been observed.

Treatment.—Iodide and grey powder; blisters. January 10 (12 days from failure of sight, six from beginning of treatment). L. counts fingers everywhere, except at centre and nasal parts of F. Retinal arteries as before, veins decidedly too large; there is now some diffuse haze of retina; vitreous n. Commencing salivation.

17th (three weeks from failure) L. 20/70, and reads 8 J., "the word itself is plain, but around it there is a sort of purple mist. At night things look pale, and people's faces look ghastly." On trial with Bull's coloured spots there was no true colour-blindness, but confusion between red and yellow, green and blue; but I did not examine the F. for insular defects. R. V. 20/30.

January 26 (four weeks) L. 20/30 and 2 J. The fundus "I should say now shows nothing unnatural"; no choroidal disease. R. 20/20 and 1 J., some large patches of exposed sclerotic below the O. D., probably an incomplete congenital insular coloboma: no disease elsewhere.

In March, 1889 (*five years later*), Mrs. G. came to St. Thomas's Hospital, thinking her eyes had failed again, but Mr. Laws found that she only needed better glasses, V. being full in each eye, and

that the fundus of the L. was (as at my own last note) normal in appearance, and F. (measured on perimeter) full.

Mrs. G.'s mother died at 53 of a "fit," having been subject to "hysterical fits," which "turned into epilepsy" since she was 40; she was not paralysed.

Mrs. G. was one of eight children, all living except one, a brother (Thomas H.) who had been under my care at St. Thomas's Hospital in 1881 for failure of both eyes, which I had diagnosed as due to tobacco, though his friends thought it was due to chlorodyne. The fact was that he was an inveterate smoker and drinker of chlorodyne. Some time after he had left me he was in an asylum for two years, where he was kept from chlorodyne but was allowed to smoke. Soon after coming out he resumed his old habits, became utterly demoralised, and died in 1884 nearly blind.

The diagnosis of retrobulbar neuritis is seldom difficult if attention be paid to the cardinal points, viz., the mode and rapidity of failure of vision; pain; the pupil; the acuteness of V. (refraction being, of course, correct or corrected); the field examined on the perimeter, but specially the central area tested for insular defect by means of coloured (red or green) spots of various sizes; the ophthalmoscopical appearances.

Retrobulbar neuritis of exceptionally rapid onset and high degree is occasionally difficult to distinguish from thrombosis of a branch of the retinal artery or from possible hæmorrhage into the sheath, until we find on careful inquiry that the sight was lost rapidly but not suddenly. It is important to distinguish the condition from congenital amblyopia of one eye, whether associated with a squint or not. Still more important is it not to confuse retrobulbar neuritis with hysterical amblyopia, an error into which we may be led by the frequency of this neuritis in neurotic women: the distinguishing marks are found in the state of the pupil and field, in the mode of onset and in the pain when present, apart from whatever the ophthalmoscope may reveal. Whilst maintaining that the disc usually shows some

inflammatory changes early in the case, I freely admit that such appearances may be absent or equivocal; and I therefore regard the investigation of the symptoms and the state of the pupil as more important than the ophthalmoscopical examination in distinguishing an early stage of retrobulbar neuritis from hysterical blindness of one eye. Another difference is in the severity of the loss of the sight; hysterical amblyopia is, I think, never slight in degree, whereas the retrobulbar disease may be of any degree from the very slightest to total blindness. In retrobulbar neuritis vision is not bettered by any assistance or suggestion; in many of the functional ("hysterical") cases the patient, though unable to see at first, improves after staring at the test types, whether the near or far set, and finally reads each successive smaller size with a jerk or a rush, especially if coaxed by false lenses, which are represented as gradually increasing in "strength."* Central scotoma is, I believe, very uncommon in hysterical cases, whilst, as already stated, it forms a constant feature in the retrobulbar disease.

The only instance in which I have seen "hysterical" amblyopia take the form of central loss of field was the following:—

Relapsing functional central Amblyopia of one eye in a boy whose other eye was defective from Central Choroiditis.

Master W. (P. 19—45) was 13 when his father first brought him to me on September 19, 1889, for recent blindness of the L. eye. The boy said that just two weeks ago at half-past ten in the morning, something came over the middle of the sight of the L. eye, got to its worst during that day and remained the same till I saw him on September 19 as above.

The condition then was:—

R. V. less than 6/60; a patch of pigmented choroidal atrophy with fibrous deposit just at Y. S. and some other patches rather further further out; O. D. clear and rather paler than L. Direct

* I mean a + and − lens of equal value superposed, beginning with + and −0·5 D. and going on to + and −1 D., &c.

reflex action of P. less good than L. All the fundus changes appeared to be old, and he had been told by an ophthalmic surgeon several months before, that this eye had probably been useless for some time.

L. V. less than 6/60; with − 0·25 D. = 6/60; not 20 J. Rough examination of F. shows a large central blind area with good periphery. P. quite normal to light, acting better than the R. O. D. of better colour and less clear than R., and retina shows watered silk reflex. My note was "probably the R. O. D. is the morbid one." The defect appears to him as a bright red cloud with a fleecy border in the sunlight, but as a yellow cloud in ordinary light. The F. measured next day, showed a large rudely circular scotoma, absolute at the centre; at the periphery of the scotoma, which about corresponded with the 30° circle, the test dot moved centripetally becomes dim, and then about 5° further in it "goes into a yellow mist" and entirely disappears. Periphery of F. to white (10 mm. spot) full and prompt.

Knee jerks very brisk.

On October 4, the field was in all respects the same and V. no better; but the P. was still quite normal to light; and the O. D. showed no decided change. In spite of the activity of the pupil I thought, in view of the disease in the other eye, that treatment for retrobulbar neuritis was advisable, and I asked Dr. Maurice Duke, who sent the patient to me, to keep him in bed and give small doses of mercury until, if possible, the gums were touched; and to use several small blisters. He took mercury four times a day for three weeks with no marked physiological effect, but at about this date (Wednesday, October 30th), the boy said "the cloud had got very thin," and on 31st, he could see well. On November 8, I found he could see 6/6 partly and 1 J. fluently, O. D. n. as before.

The previous history was to the effect that there had been some complaint of the sight the previous Christmas, for which it was thought best for him to stay away from school for a term. Then in July he had an attack of blindness of L. eye stated to be like the one above described, but only lasting a few days; and again in August he had a similar attack which came whilst he was watching his float in fishing, and got well in a few days.

He was brought again in February, 1890, as blind as ever with

the L. (the fourth attack), unable to count fingers at 6 inches, but with the P. and O. D. normal; the "cloud had grown over the eye" during one day a month previously. I did not again measure the field, but put him to bed in St. Thomas's Hospital, and made a show of treatment by the inunction of simple vaseline; he was soon well.

He was an intelligent quiet good boy, not apparently neurotic. First of all I suspected malingering, then hysteria, then after taking F. I veered towards retrobulbar neuritis for a time though there was nothing positive in favour of it. The loss of sight was of course functional in this boy's L. eye, and I suppose it took the form of central scotoma because the other eye had previously lost its central vision from organic disease.

Central localised choroido-retinitis often causes a well-defined insular defect in the field, and when the mischief comes on rapidly the diagnosis from retrobulbar neuritis may be difficult.

The distinction between an idiopathic and a symptomatic case of retro-bulbar neuritis is sometimes quite easy; in other instances, as has been indicated, it may be difficult or impossible. To recapitulate the main differences: in the symptomatic family pain, when it occurs, is often worse and more lasting than in the idiopathic cases, visible changes at the disc sometimes much delayed, and recovery of sight less hopeful; paralysis of other cranial nerves (from the first to the seventh) and signs of disease of bone at the base are fairly common in this family, whilst indications of chronic disease of brain or spinal cord—often incipient only—are far more frequent in the idiopathic family. But it must be pointed out again that the members of what I call for convenience the symptomatic family, vary much among themselves, far more than those classed as idiopathic, and it is to the idiopathic form that I ask most attention to-day.

You will see from the table* that the idiopathic family is much the larger, and for that reason, and even more because this form of disease of the optic nerve, whether lastingly

* A tabular classification drawn up for the lecture.

single or eventually double, is apt to prove a harbinger of some form of spinal sclerosis, this family is the more important form of the two. The idiopathic series or family can, without going into needless minutiæ, be subdivided into (A) a single, and (B) a double, group, and as will appear later on, the group of double cases is again divisible. The single cases (Group A) are by far the more numerous, even allowing that some of them are incorrectly classed and eventually become double. Thus in 88 cases in family I (idiopathic form), Group A (only one optic nerve affected) contains 73, Group B (both optic nerves attacked) in all its varieties, only 15.

The prognosis for sight in the whole idiopathic family is, speaking generally, good. The slighter cases often recover vision that is, for all ordinary purposes, perfect; and even in the worst examples improvement from nothing, or mere hand movement, to 6/12, more or less, is not uncommon. The final result is usually reached in from five to ten weeks from the date of failure. The following are typical single idiopathic cases:—

Rapid failure of L. to fingers at 3 feet, with scotoma and definite papillitis. Rapid improvement to almost perfect recovery with pale disc and some residual light-sense defect, in five to six weeks.

(Notes by Dr. F. A. Hayward.)

Ellen B., æt. 27, came to Moorfields on January 27, 1897. (Case 77 O. P. letter.)

She said that on 20th, when at a theatre, she noticed pain about the L. eye when looking sideways. The pain continued for about four days, but does not seem to have been severe. On 23rd she found L. eye going, and it rapidly became worse, but as atropine was used by her doctor a day or two later, the exact progress of the failure cannot be stated.

On 27th (four days after failure of V.) the L. eye could only count fingers at 3 feet; pupil dilated (from atropine). O. D. swollen 2 or 3 D.; hazy and streaky; no hæmorrhages, but veins

rather distended; refraction at Y. S. about H. 2 D., at O. D. 5 D. R. V. 6/6; H. estimated at O. D. 2 D. F. showed no peripheral loss, but a horizontally oval scotoma, from centre to blind spot, was easily found with a 10 mm. white test object.

Dr. Hayward's careful examination of the organs and the nervous system revealed nothing unnatural, and the family and personal history were negative, except a liability to sick headaches. Such a sick headache with vomiting occurred the day before she noticed the orbital pain.

Treatment.—Calomel purge: iodides and a blister 1 inch square every alternate night (three blisters in a week).

February 3 (10 or 11 days from failure, one week after admission). V. of L. is much better, 6/36 or 6/24. Appearance of O. D. as before.

February 24 (three and a half weeks). L. 6/9, but only 6/24 of Bjerrum's gray types (R. seeing 6/9 of same gray types).

March 17 (six and a half weeks). L. 6/6, 6/12 of Bjerrum's types (R. = 6/9 of Bjerrum's types). L. O. D. quite clear, uniformly pale all over, and not now at all swollen. No scotoma could now be found on the most careful trial.

Severe acute case, with slight but definite changes passing into pallor of disc. Almost perfect recovery. No field charts.

Sister K., æt. 30 (Case 79, P. 40—62), apparently in excellent health, and not overworked, or living in any way an unnatural or unhappy life, has worn glasses for H. since she was 13, and for some months past. + 2·5 D. cyl. with + 1 D. sph. for each eye ordered by Mr. Attlee. The R. eye has never been so good as the L.

She had always been liable to asthma, but this had been much better since she had left London for a country sisterhood, six months before this narrative begins; she had been somewhat constipated when she first entered the sisterhood.

It was for failure of the L. (or working) eye that she came to Moorfields, on October 24, 1896, stating that the sight had failed rapidly three days before, and that five or six days before the affection of sight there had been some pain in the L. brow and in the eye, especially when looking to one side. The pain had

already diminished before she came. Mr. Holmes Spicer found *on admission* that V. in L. eye was reduced to counting fingers in upper part of F., and was worst at centre of F. The colour of a red card 4 inches square could be recognised only in the upper part of the F. She described a strained feeling on looking to her R., but the movements were full, and there was no tenderness on pressing the eye through the eyelid. Eserine had been used before she came, and the reaction to light could therefore not be satisfactorily observed. The ophthalmoscope showed a "slight ill defined haze and swelling of the disc as compared with the R."

She was ordered to take 6 (and later 8) grains of iodide of potassium, and 10 of salicylate of sodium thrice daily.

On November 8 (two weeks after failure) she said V. was considerably better. The O. D. was paler, but still rather hazy. Direct action of p. present, but much less than that of right eye. Treatment continued.

On November 19 (four weeks). L. with her glasses 6/12 or 6/9, but only 6/36 of Bjerrum's "A" types; disc clearing and getting paler. This compared with R. (with her glasses) 6/12, and Bjerrum's "A" 6/18, or 6/12.

Thus the V. of L. for black and white had improved in the most striking manner, though the sense of light contrast, as shown by the ability to read gray types, was much behind that of the healthy, but originally somewhat defective, R. eye.

On December 10 (seven weeks). L. 6/6 just, but Bj.'s "A" only 6/18 slowly though correctly (against R. Bj.'s "A" 6/12 partly).

May 3, 1897 (six months). Objects still look "lighter" coloured to the L. than to R. eye. V. L., with a slight change in her glasses 6/9, not so well as R., Bj.'s "A" 6/18.

R., with a slight change in her glasses, 6/9 partly, Bj.'s "A" 6/12.

Thus, whilst the form sense of the affected eye recovered almost fully, the light contrast sense did not recover in quite the same proportion.

In this case Dr. Emerson reported that the organs and the functions were apparently healthy. We did not examine the knee reflexes. The patient's parents had been double first cousins;

the mother died 16 years previously, of rheumatic fever; the father was living, and subject to eczema, which was said to be gouty.

March, 1899. Condition of eyes as at last note, 6/9 with each eye with the best correction, the L. (neuritic) eye not quite so well as R.; Bj.'s. types 6/12 with each.

pid failure of left to almost total loss of perception: definite papillitis. Quick recovery of V. and subsidence of papillitis.

(Notes by Dr. F. A. HAYWARD.)

Kate F., æt. 19 (Case 126 O. P. letter), came to Moorfields on March 31, 1897, stating (1) that six weeks previously her L. eye had ached and become rather dim, but had got well in a few days; (2) that six days ago the same eye had failed again, and become blind between then and the date of coming to the hospital: this time there had been no aching or pain either in the head or about the eye. She had been exposed to cold wind when cleaning windows the day before the second failure.

On admission, L. has only p. l., cannot distinguish fingers against the light; pupil contracts a very little to the full light of the electric ophthalmoscopic lamp, but the contraction is not maintained. Some tenderness on pressing the eye back into the orbit, but no pain from movements of the eye. O. D. redder than the R., hazy, especially below, and decidedly swollen (2 D), veins rather large, but no hæmorrhages; no other changes; refraction at Y. S. Em. R. 6/6, Em., O. D. n., and much clearer than L.

No nasal symptoms. Patient seems healthy. Catamenia regular; was "anæmic" two years ago, but is not so now; no history of rheumatism. Her mother suffers from rheumatic gout, affecting the foot chiefly.

Treatment.—A blister 1 inch square to the temple every night for three nights: iodide of potassium and salicylate of sodium in moderate doses.

April 3 (10 days from failure), p. l. is decidedly better, but still requires the contrast to be strong.

April 10 (17 days). V. better, but not 6/60. Swelling of O. D. now only 1 D.

April 17 (three weeks). V. of affected eye 6/36.

April 24 (four weeks). V. of affected eye 6/12; O. D. now seen best without any + lens. No later note. No note of F. or of knee reflex.

Acute severe case. Good recovery. Notes not exhaustive.

Miss B., æt. 19 (Case 110, P. 20, 205) was sent by Dr. Watters of Stonehouse. She had always been short-sighted, and the vision of L. seems never to have been so good as the R.

On the evening of Friday, May 23, 1890, she noticed some mistiness of vision, and the next morning found the R. (or better) eye nearly blind.

When she came on June 6 (two weeks) V. of R. was fingers at 4′; the direct reflex action of p. was very little; the disc hazy, congested, and slightly swollen, no hæmorrhages, vessels normal. L. 6/24 improved to 6/18 by − 2 D. cyl. with − 1 D. sph., but result indefinite both at this and subsequent trials. The shadow test and estimation by ophthalmoscope showed M. 3 D. in vertical meridian, 1 D. horizontal.

Ordered to take iodide and mercury, to have blisters to the temple, and wear dark glasses.

On July 7 (six weeks) R. with correction 6/18.

On October 3 (five months) R. "as good as the L." she says. Careful estimation showed M. 3·5 D. vertical, and with this correction she had 6/18, not more. There are no notes of F. or colours, nor of final ophthalmoscopical appearances; and the recovery seems to have been only partial. But the general features of the case are characteristic and well marked.

A few days before the eye failed she had been travelling by railway with her face to the engine, and the window open; but she did not feel cold. The patient had never had arthritic symptoms; but her mother and mother's mother were subject to gout, and there was also gout on the father's side.

A case in which, with very little loss of V. for black and white, the patient complained of failing sight in one eye, and great loss of light-difference sense was found, together with scotoma; marked pallor of O. D.

(Notes by Mr. H. PARKER.)

Thomas J. D., æt. 21 (Case 129, O. P. letter), compositor, came to Moorfields on 2nd of October, 1897, complaining that for a week past he had not seen properly with his R. eye. The eye had been aching a good deal, and he had some toothache lately, but there had been no pain or tenderness about the eye, and no headache.

Except that the knee jerks were found to be very brisk, there was nothing to be noted; he was pale (indoor occupation), did not smoke, and denied ever having had any venereal disease; family history gave no assistance.

Though he said he saw badly with the R. eye, Mr. Parker found V. in this eye 6/9 raised to 6/5 in + 0·5 D., the L. V. being 6/6 with and without + 1·5 D.

At this stage it would have been easy to dismiss the case as indefinite, for the disc appears to have shown no decided alteration. But the pupil was observed to be rather larger than the other (*i.e.*, less active to direct light), and on then trying V. with Bjerrum's types, Mr. Parker found a very great difference between the light sense in the two eyes, the R. with correction seeing only 6/36, whilst the L. saw 6/9. There was a small scotoma just above the centre of the F. in the R.; no peripheral loss.

October 16 (two weeks later, or three weeks after failure began) the above observation was repeated with the same result, and there was now decided pallor of R. O. D. as compared with L.; no change whatever at the Y. S.

On December 18 (nearly three months) V. of R. seemed not quite so well, viz., with + 1·5 D. 6/12 Sn. and 6/60 Bj. against 6/6 of both types in L. with the same lens. The R. O. D. is noted as showing much pallor. But six weeks later (January 29, 1898, four months or more) R. again sees 6/9 Sn with + 1·5, and after this he did not come.

The case was slight from beginning to end, and apparently

slower in coming to an end than some cases are. It might easily have been overlooked had not the patient been accustomed by his trade to accurate observation, and had Mr. Parker not quickly appreciated the leading features, viz., the state of the pupil, the very marked lowering of the light sense and the gap in the field.

A similar slight case. Notes not so complete.

Mrs. D., æt. 45 (Case 88; P. 30, 157), was sent by Dr. Morison on May 4, 1894, for a defect of two weeks' duration in L. eye with a "bruised feeling at the top when the eye is moved." Is rheumatic, and has "barometric joints."

May 4, 1894. R. – 4·5 D., 6/9 well; Bj.'s "A" = 6/9.

L. – 4·5 D., 6/9 "blurry"; reads word by word, and has to look sideways to it. Bj.'s "A" = 6/24 barely. Reflex action of p. less than in R. Relative scotoma at centre of F. and above; periphery not measured. O. D. blurred; edge invisible by direct examination; no swelling.

Blisters and salicine.

Smoked glasses.

June 14. R.—4·5, 6/6 partly; Bj.'s "A" 5/6 partly.

L.—4·5, 6/6 partly, but not so well as R.; Bj.'s "A" 6/12. No ophthalmoscopic note.

Not seen again.

The following was probably a case of slight retrobulbar neuritis, but the micropsia and the retinal haze point rather to central retinitis of low degree. It at least illustrates the occasional difficulty in diagnosis already alluded to.

Mrs. Walter B., æt. 69 (Case 105; P. 22, 61), sent by Dr. Schacht on January 26, 1891, for slight defect of R. found out accidentally three weeks before; a little mist as if with dust on it, and type looks only half the size it looks to the other eye; all things look a shade darker than to the other eye; a picture with this eye looks as if in the shade; with the L. it looks as if in sunshine.

Now, R. p. and T. n. (p. rather > L.); Hm. 4 D.; V. 6/6 partly

L. Hm. 3 D.; 6/6 well.

R. (after mydriasis) O. D. and surrounding retina slight haze and decidedly different from L.; Y. S. quite n.

A well marked relative scotoma for 5 and 10 mm. red and for pale pink from fixation point outwards "exactly as in tobacco." Periphery of F. (to hand) full.

(R., no trace of scotoma).

March 16. R. w. + 4 D., 6/6 partly, and still looks "smaller" than to L.; Bj.'s "A" 6/18; O. D. certainly a little paler than L., especially on Y. S. side. Scotoma to 5 mm. pink still present, but not $> 5°$ or $10°$ each way from centre.

L. w. + 3 D., 6/6 well; Bj.'s "A" 6/9.

Of the double cases (idiopathic family Group B) it seems that we may for the present distinguish B 1,—Cases which resemble those of Group A (Single Cases), but suffer one or more subsequent attacks like the first, and affecting either the same eye or alternating between the two; and which are complicated by loss of power or loss of sensation in one or more limbs (usually the legs), that may (like the loss of sight) be temporary, but often settles into something permanent. Repeated attacks of this optic neuritis seem usually to leave some degree of permanent defect, but not necessarily a high degree.

There is no doubt that some of the Single Cases (A) are of the same essential nature as the double ones just mentioned, and may sometimes, if observed long enough, be seen to pass into the latter. At present I have notes of only some six cases of the Group B 1, and select the following:—

Miss —— (Case 93; P. 29, 251). Early in 1894, she being aged 29, the L. eye failed, and when I saw her early in March, V. was 6/60 and 15 J., the O. D. pale with some diminution in size of the arteries, and the direct reflex action of p. much less than in the R. The F. does not appear to have been examined. 12 months later (April, 1895) the L. had improved to 6/18; the O. D. was pale as before, and F., though of full expanse, showed an absolute scotoma close to the centre. The R. (hitherto good) had now been failing for a fortnight, and its V. was 6/36, with

slight haze of O. D., a small absolute scotoma but full expanse of F. Knee jerks now very brisk, but no ankle clonus.

June, 1895. R. and L. each see about 4/60 and 16 J., both O. Ds. pale; ps. act to light.

November. Thinks sight worse, and says that it varies a good deal and often; R. (2nd eye) 3/60; L. (1st eye) 6/24; Fs. about as last time. Lately some pain and weakness in L. leg "as if it would give way."

April, 1896. Reports "eyes better on the whole, but still vary; left much better, but right still very dim."

February, 1899. Her brother, a medical man, reports that about three years ago her sight again got much worse for a time but improved again, and that now, with both eyes, she can read some of 14 J., and do a little sewing, play chess, &c., but cannot see 6/60; that she cannot walk far, and has decided weakness of L. hand, arm, and leg.

Miss —— (Case 115; P. 16, 134), æt. 21, was first seen June 21, 1888, for failure of sight in the left eye which had begun four or five weeks before, and got worse for the first few days. As she had quite lately gone through a severe distress, and had several years previously more than once behaved strangely and been considered morbid and fanciful, the eye failure was thought to be hysterical both by her doctor and parents. Some numbness of the legs up to the waist which had come on when the eye failed seemed likely to be of the same nature.

June 21, 1888. Well nourished and apparently strong. Is reticent, but though professing to make light of her ailments, is really very pessimistic about her sight. Sleeps badly and reads late into the night.

With L. eye barely sees 6/60; F. shows large, dense scotoma for red; O. D. decidedly pale, especially on Y. S. side; vessels n.; no appearances of embolism; P. acts less freely than R. Decided lowering of sensation to pin-pricks in both legs; more in R. than L.

Ordered blisters to temple, and to wear smoked glasses; and sent for opinion to Dr. (now Sir William) Gowers as I could not agree that the case was hysterical. Dr. Gowers thought that the

leg symptoms might be due to a temporary affection of the cells of the posterior columns of the spinal cord, and were thus not ordinary hysteria; but that the legs would probably recover, and that practically therefore the symptoms might be called functional.

July 3. L. sight still only 6/60; F. shows no contraction; loss of perception of green in all parts; red is correctly perceived, but with hesitation, and worst close to centre of F.; blue promptly seen in all parts of F. Action of pupil to light markedly less than in other eye. O. D. as before; considerably paler than R., especially on Y. S. side, and also a degree less transparent than R., and vessel's coats rather thicker.

To take iodide and lithia and mercury (gr. 1 of tannate), and have mustard to spine.

July 27. L. has improved (from 6/60 barely) to 6/24 and 6/18 after intent looking, and can read in the hand from 10 J. (fairly) up to 2 J. (words). Glasses do not help.

September 7. L. now sees 6/12, and some of 6/9. On November 1, Dr. Gowers, who had seen her again, found a tendency to stumble when going up steps, and a little want of equilibrium with the eyes shut and feet together, and thought the development of permanent organic disease more likely.

December 11. L. sees 6/9 slowly, but well. For a week past, R. has been failing, with pain in or behind the globe. Now can but just see hand moving in the upper outer quadrant of F. P. almost motionless to light, and becomes very large when the L. is covered. O. D. red, rather hazy, and veins too full. Has of her own accord resumed iodide and mercury. To use blisters again.

December 21. R. now sees hand easily in all parts of F., except at centre and at down-in quadrant. O. D. is slightly swollen (H. 2 D. as compared with H. 0·5 D. at Y. S.).

January 22, 1889. R. sees 3/60, but not quite 6/60. O. D. still hazy, but temporal side getting pale.

February 26. R. O. D. much clearer, and Y. S. side paler. Now sees 6/60.

Was not seen again until May 17, 1889, when she came saying she was worse, and on trial, V. was very bad in both eyes. (This fresh failure was certainly recent in L. The R. might have remained unaltered since February; but I think it had

improved and failed again, for had it remained at 6/60 ever since February, the very marked improvement which now soon took place would have been unlikely to occur.)

Now (May 17) R. barely sees 6/60, F. of full size, but shows a dense scotoma from centre outwards.

L. 6/36, a similar scotoma from centre outwards but not so large as in R. F. of full size. Ps. act to light. O. Ds. about as last time. Confuses all red, green, and gray colours.

No further note until June 11, 1891 (two years), when R. sees 6/12 and L. 6/9 partly, and easily reads small print. Both O. Ds. very pale on Y. S. side, but no other changes.

The next note is July 22, 1892, when she said she had lately had a short attack of double vision "caused by the left eye"; this had now quite passed off. V. was the same as last time, not quite 6/9 with the two eyes together.

In the summer, 1893, again had double sight, chiefly when looking to her left, probably L. external rectus.

November 1, 1893 (five years after first note). Now no recognised diplopia. Print sometimes gets confused or swimming both with and without her weak + glasses; also distant objects looked at fixedly, either with both eyes or either eye singly become "unsteady," "much like rain running down a window pane." Perhaps this may be due to minute nystagmus made worse by effort. Complains a good deal of pain at back of neck (about seventh vertebra). V. same, 6/9 partly with both together The L. leg still drags somewhat, and is weak, with effort she can manage to put the foot upon the chair seat. Knee jerks (now tried by me for the first time) very brisk.

In April, 1897, the L. eye again failed rapidly with some days of pain in turning the eye about. I saw her on 12th with V. only 6/60 in this eye eccentrically (central scotoma passing also upwards from centre), P. acts less to light than R. O. Ds. as before.

In August, V. was 6/9 as before the attack. Sir William Gowers saw her again about this time and told me she had undoubtedly a sclerotic lesion of the cord, probably focal myelitis in the dorsal region.

One of this lady's elder brothers is also the subject of a form of spinal sclerosis with central blindness of one eye from partial

optic atrophy, the other eye failing slowly many years later. I have seen him at intervals from 1890.

The chief events and dates in his case were :—

1879, æt. 19. Double vision with drooping of eyelid (he does not remember which eye). Recovered in a few months without treatment.

1884. Failure of R. eye during one day with some pain, could not see his own hand with the eye. Had much advice and was thought by some to be malingering, because there was so little sign of disease in the eye. Sight improved a little but never became useful, and when I saw him in 1890 (six years after failure) at aet. 29, he could just read 16 J. with the eye by looking sideways, and there was a large loss in the F. from the centre up and out. O. D. atrophied. L. 6/6 F. full (perimeter) and no scotoma; doubtful pallor of outer side of O. D. Ps. act to light and accommodation, but R. less active to light than L. Articulation slow and catching; he walks carefully as if not at ease on his legs; under treatment for "general weakness." Denies ever having had intercourse.

In August, 1892, the eye condition was the same, and the L. continued to see 6/6 well, though its O. D. was somewhat pale. But he was getting much more ataxic and weak, articulation was more difficult, and his mental power seemed less than before.

In May, 1898, he came again, and this time the L. had sunk to 6/18 barely and 8 J. The R. still read 16 J. Ps. acted somewhat to light. Internal recti acted very imperfectly and R. diverged; no ptosis; other ocular movements full; rapid nystagmus of both. O. Ds. as before, L. much less pale than R., central vessels about normal. Incontinence of urine; constipation; speech jerking, gait jerking and weak; needs two sticks, and a servant to help him.

Several attacks of loss of sight, followed by recovery but leaving the discs pale. Various other temporary paralyses of motion and sensation. Patient under observation nearly twenty years.

Miss W. (Case 123, P. 7—161) gave me the following account :—

At the age of about 16, in 1876, after much family trouble

(money losses) she had an illness, accompanied by headache, loss of sleep and some "cramps" in limbs, during which she became nearly blind of both eyes. The late Mr. James Adams told her at Moorfields, she would soon get well, and she did, vision recovering "perfectly in both eyes" in six weeks.

Remained well, acting as a teacher, till 1880, when, at æt. 19, she again had "numbness and cramps," and the R. eye (not both eyes) again went rapidly almost blind. The eye improved and the other symptoms passed, and for about 18 months she was as usual.

During nearly the whole of 1882 she was under the care of a colleague for repeated failure of vision, alternating between the two eyes, but (as the Hospital letter showed) never very bad in both eyes at the same time. In August of this year, she came to me wishing another opinion. She was then an energetic, emotional, but very intelligent person of good frame and well nourished; no tremors, but some "numbness of the arms" (patient's statement). Complained of seeing colours badly, and of difficulty in judging the distance of objects because they looked too small. R. 6/60 and 14 J.; O. D. very pale all over, and arteries too small; P. large and sluggish to light. L. 6/12 and 1 J., O. D. pale especially on outer side, but much less so than R., and arteries not diminished; P. sluggish to light. Fs. to white full; marked confusion of red and green wools with R., and the same but less marked with L. in which, though no scotoma could be defined, colour vision was best eccentrically. Refraction Em. in both. No defect of smell nor dropping of fluid from the nostrils.

A year later (October, 1883), better in all ways and V. better (R. 6/18; L. 6/6 slowly), but said objects still looked small and far away to the R., and that long gazing either at far or near objects caused dizziness of R. and brought on headache.

On June 9, 1886 (aet. about 26) the L. (better eye) failed again rapidly and badly, but had partly recovered (to 6/12) when she came to me on the 26th. This time she also had double vision due to paresis of R. external rectus, and again suffered from headache and retching. R. 6/18 as before. She took small doses of iodide for about three months, improved in health and lost the diplopia.

On May, 10, 1890, after two or three weeks of pain first as general headache then localised in or behind the eyes, L. failed again, the pain ceasing when the sight went. On 19th, I found V. of L. only fingers at 12 inches, and direct reflex action of P. bad (worse than R.) O. D. as before, and showing no inflammatory appearance. R. as before, 6/18. V. of L. gradually improved again and by the end of July was 6/9, but became dim after a short effort. In this attack she again took iodide, and mercury was tried but caused diarrhœa.

In the late spring of 1893 after nursing her mother through a pulmonary illness, Miss W. was ill again (it was called influenza) and in June her L. leg became "numbed and felt as if tied round with a tight cord" and lost power, so that she had difficulty in going up and down stairs; it got better but then her R. arm followed in the same way so that she lost power of writing legibly, could not hold a cup and had to have her food cut up—the eyes did not suffer this time. I saw her in November, when the leg and arm had recovered, but the leg (L.) still "dragged" when she was tired and she considered her hand-writing (R. hand) still unsteady. Knee jerks extremely brisk. There were no noticeable tremors or nystagmus, but she had an anxious agitated manner and was much thinner. The discs had not changed in appearance since I first saw her, the R. being still paler than L., and V. was unaltered (R. 6/18, L. 6/9 partly); some qualitative loss in the nasal part of L. F., and to a less degree in the temporal part of R. I have not seen her again, but in 1895 she wrote, in very depressed spirits, that in spite of the recent illness and death of her mother, sight was keeping the same. She would then be about 35.

The remainder of Group B of the idiopathic family (comprising nine cases) contains some six examples of a different form which, for present convenience, I have called the Harding cases, after the name of the patient in the first case of the sort that I saw. The mark of these cases is rapid failure of one eye, shortly followed by similar failure of the other, and by quick and good recovery of both without recurrence of blindness or any nerve complications.

Case 74.—Male, æt. 23, 1883 (published as Case 11 in Trans.

Ophth. Soc., vol. iv, p. 203). R. had one or more fleeting attacks of dimness, and then failed rapidly to less than 6/60 in two or three days with much loss of F., involving centre and part of the periphery; complete recovery to 6/6 in three weeks, with some pallor of O. D. L. began to fail a week after R., went through a similar attack, with loss of F. of same character, but different in detail, and recovered perfectly in two to three weeks, also with pale O. D. Pain over the brow and pain in ocular movements preceded the failure of each eye.

Except that he was anæmic, and had lost nearly all his molars of both upper and lower sets, there was nothing to be made out as to possible cause. He had never smoked, and had been absolutely continent. Seen again 1894 (11 years) with V. 6/6 in each eye, and O. Ds. as before; in good health.

In the above case both O. Ds. showed a little haze with decided pallor, especially on the outer side, within six days of the failure of sight.

Mrs. —— æt. 30 (Case 7, Moorfields O. P. letter, 1889). Rapid failure of L.; two weeks later fingers at 2 feet, marked papillitis (swelling 2 D.); direct reflex of P. bad. Six weeks from failure 6/12; three months 6/6 partly; Bj. 6/18; four years 6/6, O. D. pale and clear.

R. failed a month after L., and in a week V. = only hand movement; papillitis as in L.; two months from onset 6/12; Bj. 6/36; four years 6/9, O. D. pale. No nerve symptoms.

A few days before the first (L.) eye failed, she had a blow by a wooden handle on the L. brow, and this was followed by a good deal of pain behind the eye. No evidence of syphilis could be obtained.

We may now attempt some conclusions as to the meaning of idiopathic inflammation of one optic nerve, or of both nerves with an interval; the seat and nature of the local disease, the prognosis, and treatment.

Sex.—Of the 88 idiopathic cases 58 were in females, only 30 in males—two females to one male.

Age.—Of 86 (out of 88) cases in which the age when the attack occurred is given (or in double and complicated cases,

the age at which the disease began), 69 were 40 years or less, only 17 being 41 or more. Further, of the 69 under the 40 years limit, 60 were between 20 and 40; only three were below puberty (taken as 15), and of these two were girls not much below that age.

If we now take the distribution of the sexes in relation to age, we find amongst the 69 cases below the 40 years limit 48 females to 21 males, or $2\frac{1}{4}$ to 1; whilst of the 17 patients above 40, the males numbered 8, the females 9—practical equality.

These figures show conclusively that this form of optic neuritis is especially a disease of the most sexually active period of life, and that during that period women are more than twice as subject to it as men.*

Apart from age and sex, we can in a certain number of the cases discover something in the patient's history that may fairly be credited with a share, predisposing or exciting, in the attack, but in many nothing is ascertained by an ordinary enquiry.

Exposure to cold after the depression caused by worry, or simply after or during physical fatigue, is noted in at least nine of my cases, and this number is no doubt below the mark.

A very definite history of gout in near relatives is given in about six of the series, and knowing the conditions under which such an enquiry is often made, we must take this number too as much less than the whole truth. But the patients giving this history were all seen in private, and it is doubtful whether the proportion is any larger than it would be in any similar series of patients of the same class. Thus, although I do not think that gout has yet been proved to act directly or indirectly as a cause of retrobulbar neuritis, the enquiry is one that ought to be pushed.

Of the infectious diseases, influenza seems to have occurred

* My whole series of cases extends over more than 20 years, and the disproportion between the females and males is found to run with tolerable uniformity through the whole period.

more often than any other shortly before the optic neuritis, and it appears to be answerable for some very severe attacks.

There was a history of typhoid fever just before the eye attack in one or two, and of some form of malaria in two or three others; four cases in all.

Syphilis was certain in two of the idiopathic series, and probable in three others; but in the two cases where it was certain, the primary disease had occurred respectively 12 and 20 years before the eye attack, and in one of these the patient gave a clear history of malarious fever a few weeks before his eye failed. I think therefore we may, for the present, conclude that syphilis is very seldom the cause of idiopathic retro-bulbar neuritis, except when it causes a gumma in or upon the optic nerve; and such cases, of which I believe I have seen several, I purposely exclude, placing them in the symptomatic family. Very different is the share taken by syphilis, whether recent or remote, in causing by one means or another a symptomatic retrobulbar neuritis. In my symptomatic family I have provisionally placed 32 cases, and syphilis was certain, or almost certain, in about 17 of these—at least one-half—the primary disease dating from a few months to many years prior to the optic neuritis.

Lastly, the history shows in a considerable number of cases of idiopathic retrobulbar neuritis a predisposition to marked functional disturbance or organic disease of the nervous system. The excessive frequency already pointed out of this optic nerve disease in women at and below middle life is only one indication of this. Not only are many of the patients themselves evidently very neurotic, but in no less than 15 of my series there either was at the time, or had previously been, loss of power or loss of sensation in one or more limbs,* whilst 13 others gave a history of chronic affections of the nervous system in the family.† There was obtained, therefore, a history of either special instability or

* Cases 23, 24, 27, 28, 70, 93, 111, 113, 114, 115, 123, 132, 134, 143, 146.

† Cases 11, 15, 26, 66, 78, 79, 84, 91, 98, 118, 122, 134, 145.

actual disease of the nervous system in connection with quite 27 of the 88 cases—nearly 1 in 3. Of the 27, 7 were male and 20 female.

Slowly progressive double optic atrophy with loss of knee jerks, or the latter with loss of the pupillary light reflex, arouse a strong suspicion of incipient tabes dorsalis (locomotor ataxy). A very large proportion of these cases occur in men, and there is a history of syphilis in a considerable number.

An attack of acute retrobulbar optic neuritis usually single, or if double with much delayed symmetry, generally ending in partial or complete recovery should, I think, always cause some anxiety as to the future onset of disseminated sclerosis or some other disease of the cord, not tabes. The great majority of these cases occur in women during the sexually active years, scarcely one-third being in men. Syphilis appears to take little, if any, share in causing the attack.

Optic atrophy in the early stages of tabes often means, I believe, that the limb phenomena will be delayed or perhaps never fully developed. Will it, perhaps, be found in the future that disseminated sclerosis beginning as single retrobulbar neuritis shows less than its usual tendency to become generalised? However this may be if only one optic nerve be attacked, there is evidently less chance of escape from the general symptoms in cases where the second optic nerve becomes affected; for in no less than 6* of my 15 double cases the patient was more or less paralysed.

It is difficult, with our present imperfect knowledge, to understand the occurrence of acute disease in a portion, apparently well-defined, of only one optic nerve, the active process lasting for a certain fairly constant period, and then passing off.

The rapidity of onset points to either inflammation or slight infiltrating hæmorrhage, and of the latter,—hæmor-

* Cases 28, 70, 93, 111, 115, 123.

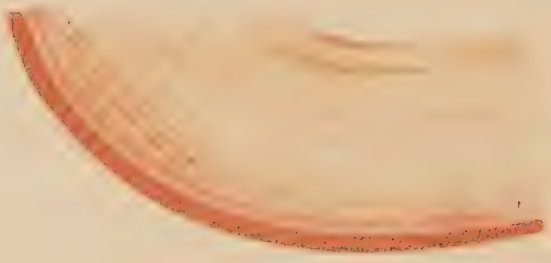
rhage from a minute intrinsic vessel in the optic nerve,—we have, I think, no clinical knowledge (the phenomena of bleeding into the sheath-space are different). The character of the pain points to the orbital part of the optic nerve as the seat of the inflammation and the frequency of early—however slight—inflammatory appearances at the disc can best be explained by a lesion near to the eye. The almost constant involvement of the central, or para-central part of the field indicates disease of those nerve fibres that come from the yellow-spot region and that lie on the temporal side of the nerve near the eyeball and in its axis further back. The scotoma often spreads out in a fan shape from the centre of the field towards or up to some portion of the circumference,—not the same sector of the field in all cases;* this seems to show that the morbid process tends to affect a wedge-shaped portion of the nerve, the apex of the wedge being at the macular fibres, the base at some (not always the same) part of the periphery.

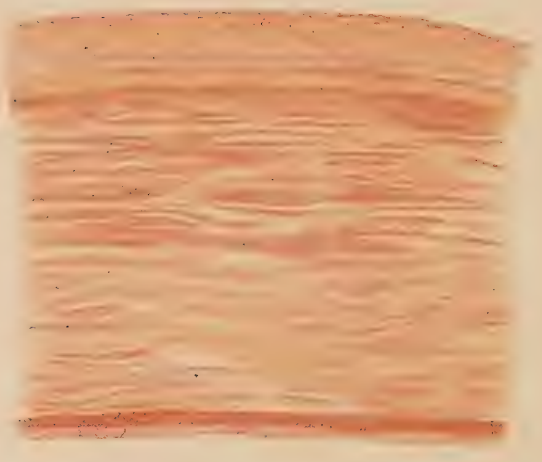
If we suppose, with Mr. Marcus Gunn, that the arterial twig supplied by the central artery to the macular fibres in some way dominates the special liability of these fibres, we cannot, it would seem, make the same vessel responsible for the varying wedge of disease. And yet if we accept as probable that some (perhaps not always the same) morbid state of the blood furnishes the foundation of the attack, the a-symmetry of the disease would be explicable only by supposing that the poison acted on the nerve fibres indirectly, inducing changes in the walls, or inflammatory obstruction in the channel of certain minute blood-vessels or lymphatics in the nerve. Speculation apart we need not refuse to consider the fundamentally toxic origin of retrobulbar neuritis simply because of its a-symmetry; for gout is often a-symmetrical in single attacks, and this not only when perhaps

* The direction of the sector of loss in the field is, according to my observations, usually outwards (up-out, down-out, or horizontal); but in a certain number the base of the sector has been inwards and in a few directly upwards.

located by injury or pressure, but in the iris and sclerotic where no such influences have occurred; again herpes zoster though almost always one-sided, is sometimes caused by arsenic taken internally.

In the treatment of retrobulbar neuritis, when seen early, I believe in the usefulness of counter-irritation and the administration of iodides and often of mercury. But we know too little as yet of the natural history of the disease, to speak strongly about the efficacy of any particular treatment, and allowance must be made, in the present state of knowledge, for apparent differences in the cause. Nevertheless it is true that most of the severe cases that have made a good recovery in my hands have done so under vigorous treatment.





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A NOTE ON THE ELASTIC TISSUES OF THE EYEBALL, AS SHOWN BY SECTIONS STAINED WITH ACID ORCEIN.

By E. Treacher Collins,

Assistant Surgeon to the Hospital.

The following description of the elastic tissue of the human eye is based upon the examination of a series of sections stained with acid orcein.

This method of staining has been introduced and elaborated by Unna, for demonstrating the elastic tissue in the skin (Maratsb. f. prakt. Dermatol, vol. xix, 1894, p. 397; Zeit. f. Wiss. Mik., vol. xii, 2, 1895, p. 240; Lee's Microtomist's Vade Mecum, 4th ed., p. 451).

In sections of the eye the tissues of which it is composed are of such varying density that they have to be treated in different ways for the elastic tissue to be shown in all parts. The length of time which suffices for staining elastic fibres in a soft tissue like the skin is not sufficient to show them in such tough tissue as the cornea and sclerotic. The length of time which is most suitable for the sclerotic and cornea is too long for the uveal tract. If the elastic fibres of the former are well shown, the latter will be much overstained, and if the elastic tissue in the latter is just right, that in the former is hardly picked out at all.

It does not, therefore, seem possible by this method to get one section of the eye showing the elastic tissue in all parts well stained.

In the cornea, Descemet's membrane stains much deeper than any other part, and in sections which have been allowed to soak for some considerable time (24 hours), it can be made to stand out a reddish-brown colour, the substantia propria remaining a dull pink. (Pl. A, Fig. 2.)

Bowman's membrane stains precisely the same colour as the substantia propria, so that this method of staining adds additional evidence to that which has been previously

brought forward as to the difference in character between Bowman's and Descemet's membranes, and as to the similarity in character of Bowman's membrane and the substantia propria.

It further emphasises the inappropriateness of the term anterior elastic lamina, as applied to Bowman's membrane.

The fibres of the ligamentum pectinatum, into which Descemet's membrane divides up, stain deeply with the acid orcein like Descemet's membrane itself. Its outermost fibres which pass into the sclerotic can be traced a long distance backwards, further than would be expected from other methods of staining, some way beyond the termination of the space of Fontana. As they proceed backwards they expand, so as to extend ultimately through about half the

Diagrammatic section of an eye. The elastic tissue staining with acid orcein being marked in red.

width of the sclerotic, having between them bundles of white fibrous tissue, equatorially and meridionally arranged.

This method of staining picks out in the sclerotic a number of delicate elastic fibres, finer than those usually met with in the skin. In sections these are seen cut in various directions, those cut transversely appearing as little dark red dots. Those cut longitudinally for the most part run parallel with the bundles of white fibrous tissue, and have a wavy course.

The outer layers of the sclerotic contain more elastic fibres than the inner, and they are more numerous posteriorly than anteriorly. Around the entrance of the optic nerve there is quite a dense plexus of them.

The lamina cribrosa is largely composed of elastic fibres, which extend across the nerve fibres from the plexus in the sclerotic on each side. From the lamina cribrosa a delicate network of elastic fibres extends backwards, around the central blood vessels of the optic nerve.

In both the pial and dural sheaths of the optic nerve there are numerous elastic fibres. In the former they are like those in the sclerotic, fine and delicate, but run a more tortuous course. In the dural sheath the elastic fibres are thicker than those in the sclerotic, but have the same wavy character, and run parallel to the bundles of white fibrous tissue.

In the tendons of the recti muscles where they are inserted into the sclerotic, thick elastic fibres are deeply stained by the acid orcein, they are especially numerous in the sheath around the muscles.

This arrangement of elastic fibres in the sclerotic which I have described, corresponds, I find, very closely with what was demonstrated by Professor Satler, at the 25th Ophthalmological Congress, at Heidelberg, in 1896. He employed Spaltenholz's method of staining, which shows up the elastic fibres black against a yellow background.

In the ocular conjunctiva many stout elastic fibres are seen lying in the tissue, between the epithelium and the sclerotic.

The elastic tissue in the inner coats of the blood vessels is picked out by the acid orcein in all parts of the eye. In the iris this elastic membrane in the blood vessels is the only elastic tissue shown.

In the choroid and ciliary body the membrane of Bruch stains deeply. I have also found the nodules of hyaline substance so often met with in pathological specimens on the inner surface of this membrane, stain in the same way.

The capsule of the lens, like Descemet's membrane, can be made to stand out a reddish-brown colour, while the lens fibres, like the substantia propria of the cornea, remain a dull pink. (Plate A, Fig. 1.)

The fibres of the suspensory ligament also stain deeper with the acid orcein than the surrounding tissue.

PLATE A.

FIG. 1.—A portion of the crystalline lens and its capsule, showing the latter deeply stained with acid orcein.

FIG. 2.—A section of the cornea, showing Descemet's membrane deeply stained with acid orcein. Bowman's membrane remaining the same colour as the substantia propria.

REPORT ON AN EYE HAVING SOME UNUSUAL CONGENITAL PECULIARITIES.

By C. Devereux Marshall, Curator.

The subject of this report was a man, William M., aged 32, who was first seen in 1887 with extensive detachment of the left retina, colobomata of both irides, and a large coloboma of the right choroid which could be seen with the ophthalmoscope, while the choroid on the left side was not visible.

He attended again in June, 1898, having for the previous five months suffered a good deal of pain in the left eye, which, as it was blind and appeared to be affecting the right eye, was excised. Patient had always been short-sighted, and the left eye was never so strong as the right, the vision of which with Sph. −11 D., Cyl. −4 D., Ax. Vert. 6/24. The left eye had been quite blind for about 12 years.

The eye was removed and hardened in formol, and the following condition was noted :—

The cornea is hazy, the iris discoloured, and the pupil blocked. There is a congenital coloboma of the iris downwards, and there are severe inflammatory changes of old standing. The eye was frozen and opened equatorially.

The pupil is much drawn towards the coloboma, and there is no opening in what is now the centre of the iris. The angle of the anterior chamber is completely closed by adhesion of the iris to the periphery of the cornea. The lens is *in situ*.

The retina is entirely detached except on one side where it forms a dense folded membrane reaching from the ora serrata to the optic disc. In it numerous cysts are developed. The folds of the retina enclose a small space which is the remains of the vitreous chamber. Both this and the sub-

retinal space are filled with coagulated albuminous material containing much blood.

There is an enormous coloboma of the choroid which occupies fully one half of the fundus. (Fig. 1.)

It involves the optic nerve, the head of which is deeply cupped. The whole of the sclerotic corresponding to the coloboma is thinned and bulged, owing doubtless to the tension under which the eye has for some considerable time been subjected.

The microscopical examination of the coloboma is interesting. The choroid can be traced running along the sclerotic in its usual position over a considerable area. It is thin and atrophic, and the pigment is irregularly scattered through its substance, showing that some inflammatory change has taken place.

As one approaches the coloboma the inflammatory changes become more marked and some hyaline bodies are seen (Fig. 2*b*), these are covered with hexagonal pigment; after this the pigment undergoes proliferation and is thicker and more distributed through the substance of the choroid. On coming nearer the coloboma the choroid is seen to cease, but a few processes extend forwards and are then lost in the tissue of the retina and sclerotic (Figs. 2*c* and 3*e*); a similar arrangement is seen on the opposite side of the coloboma.

The retina is greatly altered both in its disposition and also in its minute structure by inflammatory changes, so that some careful study is needed in order to find out its exact relations.

It appears, however, that it is entirely occupied in enclosing a space which is the remains of the vitreous chamber (Fig. 1*c*) but unlike most extensive detachments this one can hardly be termed complete, owing to the fact that the portion of the coloboma which is included within the remains of the vitreous chamber is actually lined with retina, which is in an extremely degenerate state and firmly adherent to the sclerotic (Fig. 3*a*).

The detached portion of the retina is very considerably

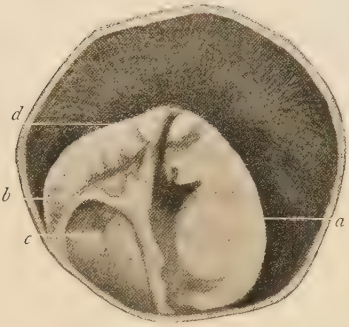

FIG. 1.

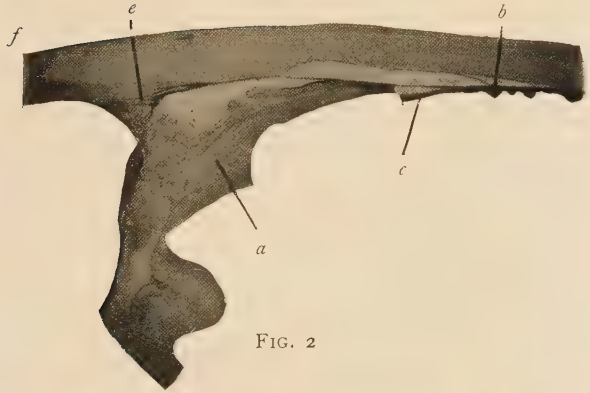

FIG. 2

FIG. 3.

From photographs by the Author. *Engraved by Bale & Danielsson, Ltd.*

atrophied and far more fibrous than normal (Fig. 3*b*), while many of the minute elements which characterise it are either altogether absent, or else are so altered as to be unrecognizable.

The hexagonal pigment layer is almost entirely attached to the choroid, but in many parts of the retina isolated cells or groups of cells are adherent to it.

That portion of the retina which is attached to the sclerotic is chiefly characterised by its extremely honeycombed appearance, it is riddled throughout with small cysts which are filled with coagulated fibrinous material which stains slightly with eosin (Fig. 3*a*). The rest of the retina is in a somewhat similar condition, but the cystic spaces are nothing like so numerous.

On that portion of the retina forming one of the side walls of the remains of the vitreous chamber, the internal limiting membrane is completely stripped off, thus forming the inner boundary of a cystic space, the outer wall of which is formed by the rest of the retina.

In the portion of the vitreous chamber near the sclerotic, there is a good deal of organised exudation as well as numerous small round cells, while further away from the sclera it is filled with material similar to that which fills the smaller cyst-like spaces mentioned above.

As we trace the retina round we soon meet with another large cyst, which is apparently developed in the substance of the retina, but much nearer the outer than the inner side. Owing to the very degenerate state of the retina it is difficult to be sure of its exact position, but it is probably between the outer nuclear and outer molecular layers. This space is partially filled with exudation.

The anterior part of the eye shows still more remarkable changes. These are illustrated in part in Fig. 4.

In order to show this, the following dissection was performed:—

The eye, as described above, had been divided by an equatorial section. The cornea and sclerotic were removed from

the anterior half, leaving the iris, ciliary body, and lens behind.

In order to see the suspensory ligament the iris which had a coloboma was removed, and thus we are able to see the ciliary processes from the front.

The position occupied by the coloboma is well marked (Fig. 4*b*), for at that point the circle usually formed by the ciliary body is incomplete, and instead of being round it assumes a pyriform shape.

From this point a median raphé runs downwards to the apex of the coloboma where the choroid on each side fails to meet (Fig. 4*c*), thus forming a uniting band between the iritic and choroidal colobomata. On cutting sections through this raphé microscopical examination shows that these are ciliary processes which, instead of uniting in the ordinary way, have met together and become fused. At the lower end of the bridge they cease, while at their upper end they are continuous with the ciliary processes which are in their proper place.

In order to produce this state of things we must have a larger ciliary body than normal, for the eye is fully developed as regards size, and is, in fact, myopic and can therefore by no means be considered smaller than normal which might, if the ciliary body only were developed, account for the apparent over-growth of the ciliary processes.

The tissue which fills up the space between these abnormally placed ciliary processes is unstriped muscle, just as is seen under normal conditions, except that its own cells have undergone proliferation and there are obvious signs of past cyclitis. There are a large number of vessels developed in this part (Fig. 5).

It is unfortunate that the eye was so altered by inflammatory changes as to make the anatomy a little obscure especially with regard to the lens.

This, as has been previously stated, was calcareous and somewhat misshapen, so that one could say but little regarding the suspensory ligament. It is probable that the lens

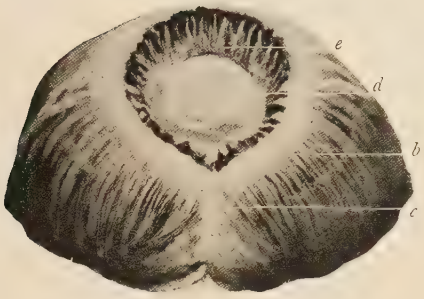

FIG. 4.

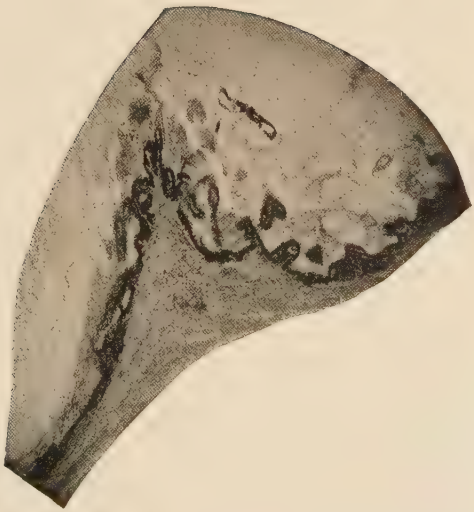

FIG. 5.

From photographs by the Author. *Engraved by Bale & Danielsson, Ltd.*

had a notch in it such as is frequently seen in these cases ophthalmoscopically, and it would have been extremely interesting to have examined the ligament and lens in detail, but this was impossible in the specimen.

As far as one could tell, the eyes had similar congential peculiarities, although the secondary inflammatory changes were far more marked in the left eye than in the right. In addition the retina of this eye is detached while the other is *in situ*.

The explanation of this condition is similar to that given for the ordinary colobomata of the iris and choroid, but in this case the developmental processes underwent some modification which lead to the defect in the choroid and iris, and possibly of the suspensory ligament and lens, as well as to an over-development of the ciliary body leading to the formation of the raphé where the processes from each side met.

A CASE OF TRAUMATIC PULSATING EXOPHTHALMOS; LIGATION OF THE RIGHT COMMON CAROTID ARTERY; PARTIAL CURE; REMARKS.

By Arnold Lawson, F.R.C.S.Eng.

I am indebted to Mr. John Tweedy and Mr. Henry Morris for their kind permission to publish my notes of this case.

Mrs. H——, æt. 41, a stout, healthy-looking woman, came to Mr. J. Tweedy's clinic, at the Royal London Ophthalmic Hospital, on March 16, 1897, complaining of severe headache and noises in the head.

Her history is as follows:—Eighteen months previously she had a blow on the back of her head, on the right side, the result of a fall downstairs. The blow was of sufficient force to render her unconscious for an hour. The accident occurred in the evening, and the following morning on waking up she found that she saw double. She went to Guy's Hospital, where she was admitted to Mr. Brailey's clinic. She then complained of occipital and frontal headache and double vision. The right eye was strongly converged, and could not be abducted beyond the midline, indicating paralysis of the 6th nerve.

R. V. = < 6/60. No improvement with glasses.
L. V. = 6/24 $\bar{c}$ - 4·5 D. Sph. = 6/9.

She was ordered a plane ground glass for the right eye, and sph. – 3·5 D. for the left, and a mixture containing Pot. iod. and liq. hydrarg. perchlor. ordered three times a day.

She improved under this treatment, lost her headaches, and at her last visit to Guy's Hospital, nearly five months after the accident, it was noted that binocular vision had returned. This was on December 31, 1895.

A few weeks later she began for the first time to experience noises in her head, at first of a trifling character but constant, and increased on exertion. These noises grew gradually louder and louder, and towards the end of 1896 she noticed that the right side of her face was flushed, and that the right eye seemed more prominent than the left. The headaches began to return

about this time, and for the last three months before her first visit to Moorfields both noises and headaches had become very rapidly aggravated.

On examination the right side of the face was seen to be deeply flushed, especially at the upper part. The ocular conjunctiva was somewhat suffused, and was coursed by several tortuous dilated veins. There was marked proptosis of the right globe, but no paralysis of any of the ocular muscles. With the ophthalmoscope the retinal veins were seen to be enormously swollen and tortuous. No marked change was noted in the arteries; but the disc was blurred, and there were patches of effusion about the veins.

R. V. = < 6/60, c̄ – 3 D. = 6/24. She had three diopters of myopic astigmatism, but no cylindrical correction improved her vision further. On placing the fingers in the hollow between the upper lid and orbital arch, the usual space was felt to be partially filled by a soft ill-defined pulsating mass, easily compressible, and imparting a marked rough thrill to the fingers. The pulsation and thrill were most marked in the neighbourhood of the supra-orbital notch, from which an enormously swollen pulsating supra-orbital vein could be easily traced over the forehead. The soft mass behind the lid was also evidently due to swollen pulsating veins. Another very large pulsating vein could also be seen running transversely outwards from the external canthus to join the anterior temporal vein. With the stethoscope a loud, rough, whizzing bruit was heard over the pulsating areas, but not elsewhere over the head. No pulsation was felt in the globe, and no pulsation was seen in the retinal veins. Pressure upon the carotid of the right side at once completely controlled pulsation and thrill.

The patient was ordered to rest and Pot. iod. grs. iij was ordered three times daily. She followed out the treatment for about six weeks, but grew much worse, and when seen at the end of that time she was evidently in great pain, and described the noises in her head as resembling the beating of a sledge-hammer. All the above-mentioned symptoms were pronouncedly worse, and it was decided to send her at once to the Middlesex Hospital, where she was admitted under the care of Mr. Henry Morris on April 30, 1897.

She was kept in bed for a fortnight to try what absolute rest in the horizontal position might effect, with a small ice-bag applied to the temple, a light diet, and Pot. iod. in small doses. She, however, got steadily worse, and her general health began to fail from the constant pain and want of sleep. About this time I again examined the fundus, and noted that there was a considerable amount of optic neuritis, and that the tortuosity had now extended to the arteries, especially to the smaller ones. A minute venous hæmorrhage was also apparent.

At the end of the fortnight further delay was obviously useless, so the patient was anæsthetised, and the right common carotid ligatured, opposite the cricoid cartilage.

Immediate and profound relief was experienced. On the morning after the operation all pain and noises in the head had left her, and all pulsation, bruit, and thrill had disappeared. From this time to the date of her leaving the Middlesex Hospital, June 17, the patient made an excellent and uninterrupted recovery. Three days after the operation I again examined the fundus, and found that a small hæmorrhage had occurred into the vitreous, but the veins were distinctly less congested, and the tortuosity of the arteries scarcely visible. The patient was kept in the horizontal position, with the head between sand-bags for rather more than three weeks after the operation. She was then allowed to sit up, and was finally allowed to move gently about the ward four weeks after the operation.

The following was her condition on leaving the hospital five weeks after the operation.

All subjective symptoms disappeared the day after the operation, and have not returned. The flushing of the right side of the face is scarcely noticeable; the proptosis and fullness under the orbital arch seem to have entirely disappeared, though there is a slight doughy resistance still to be felt. The dilated transverse vein, which was so prominent before the operation, has shrunk to a third of its former size, and the same applies to the supra-orbital vein. Pulsation and thrill have completely disappeared. The fundus is still rather obscured by vitreous opacities, but all neuritis has disappeared, and the edges of the disc look somewhat ragged and uneven. The tortuosity of the veins remains very marked, but they are much smaller and less congested. The arteries

appear quite normal. The only complication that ensued during convalescence was a slight attack of epistaxis, which occurred 10 days after the operation, and did not require any special treatment.

It will thus be seen that when the patient left the hospital the cure seemed complete.

I next saw the patient on July 7, three weeks after her discharge from the Middlesex Hospital. She then presented an almost complete relapse. All the old symptoms had re-asserted themselves with renewed vigour, with the exception of the noises in the head, which were only present in a modified degree. Since her discharge she had had two very severe attacks of epistaxis, and after each attack she felt considerably relieved for a while. She stated that as soon as she left the hospital and began to undertake home duties once more the old symptoms began at once to show themselves again. She was ordered to rest as much as possible, and to keep a piece of lint wet with lead lotion constantly applied.

I saw her again a week later. She had had another severe attack of epistaxis, two days after her last visit. This attack lasted about three hours, and since then she had felt much better. Her condition had once more considerably improved; no thrill could be felt, and the pulsation was not so heaving as before. The improvement, however, quickly disappeared, and on September 7 she was almost if not quite as bad as before ligature of the carotid. She stated, however, that she was feeling much better than during the previous week, when she had yet another attack of epistaxis more severe than any preceding it, and which had lasted almost continuously for two days. Nevertheless her condition seemed so grave about this time that the propriety of further operative measures was seriously considered; but Mr. Morris decided to wait a little longer.

However, since the date of the last attack of epistaxis, August 31, 1897, her condition has been one of steady improvement without any more relapses, and no further attack of epistaxis.

The matter may be briefly summed up by stating her present condition in October, 1898, when I last saw her.

There is complete absence of all subjective symptoms, headaches, noises in the head, &c. There is a small ill-defined swelling under the inner portion of the orbital arch, which yields a

faint expansile pulsation. No thrill can be felt, but there is a distinct bruit, though slight in nature. The right eye is slightly more prominent than its fellows, and the conjunctiva is rather congested. Pulsation can still be felt in the temporal vein above-mentioned and the supra-orbital vein, and both veins are a good deal enlarged. The right side of the face is still rather flushed. Ophthalmoscopic examination shows a very tortuous condition of the retinal veins, which, however, are nearly normal in size.

R. V. $\bar{c}$ – 4 D. sph. = 6/24. Best improvement.
L. V. $\bar{c}$ – 4·5 D. sph. = 6/9. „ „

Her general health is satisfactory, and she suffers no inconvenience, and were it not that there is practical evidence of the existence of the old trouble, which seems to present the possibility of a relapse at some future date, one would feel quite satisfied as to the result obtained.

The diagnosis of traumatic pulsating exophthalmos has now been placed upon a fairly certain basis. So that with our present knowledge, there can be no doubt that the case now recorded is one of arterio-venous communication at or near the cavernous sinus. Altogether the result of eight autopsies have been published. Six are collected by Sattler (Graefe-Sœmisch, Band vi), one is recorded by Prichard (Bristol Med. Chir. Journal, vol. viii, 115), and one by Stuelp (Arch. of Ophth., vol. xxvi, 4). In six of these autopsies a rupture between the internal carotid and the cavernous sinus was definitely proved; in one case the presence or not of a rupture remained uncertain, whilst in one case only thrombosis of the cavernous and surrounding sinuses and ophthalmic vein was found. The last-mentioned case raises the question as to how far the train of symptoms may be due to simple thrombosis of venous sinuses. There are only three symptoms characteristic of arterial rupture, viz., (1) the sledge-hammer noises in the head; (2) the peculiar whizzing bruit; and (3) pulsation which is synchronous with the carotid pulse. All other symptoms, though compatible with arterio-venous communication, might be produced by

venous thrombosis alone. Even pulsation both ocular and extra-ocular might be evident, but such pulsation would *not* be synchronous with the carotid pulse.

The prognosis of these traumatic cases if left to themselves is still very uncertain; because the symptoms produced are of such a severe character that the patient nearly always applies for relief and is subjected to treatment. It is not fair to regard the matter from the standpoint of cases of idiopathic origin among whom a few cases of spontaneous cure have been reported, whilst others have continued in *statu quo* for years without treatment. The element of trauma of uncertain quality and quantity must greatly complicate the prognosis, and the call for active treatment often becomes much more urgent than in idiopathic cases. Speaking generally, the seriousness of the prognosis might perhaps be measured by the nature of the injury and the time that has elapsed between the injury and the origin of the symptoms of arterio-venous communication. When this period is very short, a few hours or a day or two, the injury has probably been very severe and possibly caused serious damage elsewhere; whereas if the symptoms, as in the present case, have not supervened until some months after the accident, we have only to fear the results of the arterio-venous communication, and the element of trauma may be practically discarded.

As regards symptoms this case presents two of some note.

1. An interesting feature is the attacks of profuse epistaxis. Similar attacks have occurred in other cases; but not nearly so commonly as one would expect, considering the free communication that exists between the nasal veins and the ophthalmic vein. In this case each attack has been attended with profound relief, and indeed her permanent steady improvement during the last 13 months has dated from the occurrence of her last and most severe attack. This may be accounted for by the temporary lowering of the blood pressure from the loss of blood, and the increase in its

coagulability from the same cause; both of which would favour the formation of a resistent clot. At the same time such attacks are not unattended by danger, as at least one fatal case of epistaxis in pulsating exophthalmos has been recorded.

2. The irregularity in the number of nerves implicated in these cases is interesting. In the present case only one nerve, the sixth, was involved; but in many, other nerves have also suffered, and in a few, total ophthalmoplegia interna and externa has been recorded. As would be expected from its position, the sixth nerve is the one most usually subjected to pressure. The escape of the third, fourth, and first division of the fifth nerve is probably due to their position on the outer side of the sinus where the bony wall is deficient, and they might be pushed aside. In a few cases the facial nerve has been paralysed, but this must have been the result of involvement of the nerve in a fracture of the base of the skull and could have no connection in any way with the arterio-venous communication in the cavernous sinus.

Lastly, a few words as to the radical treatment of traumatic pulsating exophthalmos.

Let us look at the statistics of ligature of the common carotid. Stuelp (Arch. of Ophth., vol. xxvi, 4, 97) has brought the list of recorded cases of ligature of the common carotid for this disease to 54. I have managed to collect seven further cases, including the present one, bringing the total to 61. Unfortunately, as Stuelp points out, the number is still too small to make accurate deductions, but there are one or two interesting points. On examination of the reports of these cases we are at first struck with the immense value of the treatment and the number of *cures* which it has effected, amounting to 33 or 54 per cent. On closer examination, however, the statistics will in measure lose their value; for, unfortunately, many cases reported as cures cannot strictly be regarded as such on account of the short time that has elapsed between the report and the cessation of treatment. Thus, as one example among many, in one case reported

in Sattler's list (Graefe-Sœmisch, Band vi) as a cure, only three weeks had passed since the ligature of the artery, and the last time that the patient was seen. Further, a certain number of cases are reported as cures which would be better classified under the heading of "partial cures," for some symptoms such as pulsation and bruit have persisted, and the patient, in that he was able to resume his former occupation, reported as cured. I venture to think that no case can be considered as cured until at least one year has elapsed since the time of operation; such time, in fact, as would give one reasonably sure grounds for considering the occlusion between artery and vein to be permanent; and further, that as long as any symptom, due to the persistence of such communication, such as bruit, &c., is evident even in a diminished degree, the disease can only be considered to be held in check and not cured. Examined by this light the records of ligature of the common carotid are not nearly so satisfactory as they at first appear. In Sattler's collection, 38 cases of ligature for traumatic rupture are reported, of which 16 are noted as complete successes. Of these 16 cases the report ends in three weeks, four weeks, six weeks, three months, four months, five months, and nine months respectively, whilst in three cases the date at which the patient is last seen is not mentioned. So that out of the so-called 16 complete successes the result may be said to be in complete doubt in 10, or nearly two-thirds of the total 16. Of the remaining 22 cases complete failure resulted in five cases; death, as a result of the operation, occurred in two cases, whilst improvement to varying extent was noted in 15 cases.

Ligature of the common carotid for this disease has been practically the one and only operative measure employed since Travers tied the artery for a case of idiopathic origin in 1805. In only two cases can I find a record of any other operative procedure and both were cases of idiopathic origin. In one case, Bronner (Lancet, 95, i, 1112) tied the internal carotid with successful result; whilst in the second case

Noyes (Trans. Am. Ophth. Soc., N. Y., vol. iii) performed a successful operation, which consisted in the ligature of all the large vessels in the neighbourhood of the swelling. One can only imagine that surgeons have been and still are perfectly satisfied that there is no better method than ligature of the common carotid; but in view of the large number of only partial successes, it might be well worth while to try a modification of this treatment in the shape of ligature of the internal carotid, which, I believe, might be attended with better results than ligature of the common carotid for the following reasons:—

1. The collateral circulation after ligature of the common carotid is maintained by three channels. Firstly and chiefly by the vertebrals and basilar through the circle of Willis: secondly, by the anterior communicating artery through the circle of Willis, and thirdly by a reflux of blood through the external carotid which has its circulation quickly re-established by free anastomoses across the neck. Very little stress is laid upon this latter channel in text-books, and indeed in some it is not even mentioned; but in these cases of pulsating exophthalmos, I believe it to be of great importance, because it is the only means by which the blood stream can be re-established in the normal forward direction between the origin of the vessel and the site of injury, with the exception of one or two unimportant communications about the tympanum and carotid canal. This reflux of blood through the external carotid must also be considerable, and probably established as quickly as any other channel of collateral circulation. If the internal carotid were ligatured, this means of collateral circulation will be cut off and thereby most, if not all of the direct arterial flow through the cavernous sinus. Moreover, any stream through the artery at this point would be produced by a reflux from its cerebral branches, and would be moving in a direction corresponding to the venous current; a condition which would be more favourable to the formation of clot between the injured artery and the venous channel than

would be the case if the arterial stream flowed in its usual direction.

2. If the common carotid is ligatured, a very large amount of blood is suddenly thrown back upon the other main channels of the head and neck. If the internal carotid only is blocked, the amount of blood thus thrown back will not be nearly so great, and the necessary dilation of collateral channels to restore the circulation will probably take place more gradually, which will favour the formation of a firmer clot at the site of injury.

3. On the ground that it is inexpedient to perform a major operation when a minor one will suffice, ligature of the internal carotid should be preferred to that of the parent trunk.

4. The technique of ligature of the internal carotid does not offer any special difficulty, as at its origin it is comparatively superficial, nor is there any greater danger attending its occlusion than a ligature applied to the common carotid.

Ligature of the internal carotid might also be supplemented by ligaturing the enlarged pulsating veins around the orbit, which are so commonly seen in these cases.

Finally, this case points out the necessity for prolonged rest in the horizontal position after ligation has been performed. In the case now recorded this treatment was most strictly carried out for nearly four weeks, and yet the subsequent history of the case seems to show that another fortnight in bed would have been well spent.

Ligature of the Common Carotid Artery for Traumatic Pulsating Exophthalmos. Cases not hitherto included in Statistics. Total collected cases reported by Stuelp (Arch. Ophth., vol. xxvi, p. 4) = 54 cases.

No.	Author.	Year.	Journal.	Sex.	Age.	Trauma.	Operation.	Result.
1	Walker	1888	Lancet, vol. xciv, p. 1	M.	37	Fall off horse	Lig. of right carotid	Cured with preservation of sight.
2	Prichard	1890	Bristol Med. Chir. Journal, vol. viii, p. 115	M.	30	Fall on back of head	Lig. of left carotid ..	Improved. Subsequent death one year later from rupture into ventricles. P.M. showed arterio-venous aneurism in cavernous sinus.
3	Puzey	1893	Lancet, vol. xciv, p. 1	F.	42	Fall into cellar.....	Lig. of right carotid	Cured with loss of sight.
4	Walker..	1894	Lancet, vol. xciv, p. 1	F.	33	Blow on left ear	Lig. of left carotid ..	Cured with preservation of sight.
5	Kibbe	1897	Arch. of Ophth., vol. xxvi, p. 1	M.	—	Head crushed between two heavy logs	Lig. of right carotid	Much improved.
6	Schimanowsky..	1897	Wjert. Ophth., vol. xix, p. 1	F.	52	Fall on back of head	Lig. of left carotid ..	Improved.
7	Lawson	1898	—	F.	41	Fall on back of head	Lig. of right carotid	Much improved.

SARCOMA OF THE UVEAL TRACT.

By C. Devereux Marshall, Curator.

In a paper published in the Royal London Ophthalmic Hospital Reports, vol. xiii, Pt. ii, in December, 1891, by Messrs. Lawford and Collins, all the cases of sarcoma of the uveal tract which could be obtained from the records of the hospital were tabulated and analysed. Records of 103 cases were found, and carefully worked out so as "to obtain evidence regarding the important question of prognosis in this affection."

This paper has proved to be of the greatest service to workers at this subject; but if such evidence is to be kept up to date, it is obviously the duty of those who have the opportunity of doing so, to continue to publish reports of the more recent cases, in addition also to still further following up those already published.

In order to fulfil these conditions, the following plan has been adopted:—

(1) All the patients who have been treated or examined at the hospital suffering from this disease since 1891, have been written to, unless evidence has already been obtained of their death. Of these there is a total number of 58.

(2) All the patients who were reported as being alive in 1891, and who were included in the previous paper have been written to, and in a large number satisfactory replies have been obtained either from them or their friends, and in many cases which have during the interval terminated fatally, the cause of death has been ascertained.

(3) An attempt has been made to search the death registers at Somerset House, for the purpose of ascertaining whether the names of those persons from whom no reply has been obtained appear therein, but owing to the extreme difficulty in locating the districts in which such people lived at the time of their death, and owing also to

the restrictions put upon those carrying out what is termed "a general search" by the authorities, together with the probability of obtaining unsatisfactory results, especially with regard to the cause of death in those traced, it has been decided, on the advice of a competent authority, to abandon the search as being, under existing circumstances, impracticable.

(4) The further information concerning those recorded in the previous paper will first be stated, followed by an analysis of the 58 cases, which form the basis of this present report.

(5) All particulars relating to cases in the previous paper which are not to be found here, will be seen on reference to the original. Those which have occurred since 1891 will be dealt with in detail.

After-History of the Cases reported in the Previous Paper.

Of the 103 cases in the 1st series, 40 were dead in 1891, and since then we have found that nine others are dead, and two besides these had, when last heard of, recurrence, so that we have now 49 cases in which we know that death has occurred, and if we include the two other cases, this will make 51, besides 46 others which are unable to be traced.

In 1891 the following after-history of cases was obtained:—40 or 50·63 per cent. were dead; 39 or 49·36 per cent. were alive, and in the remaining 24 cases the after-history was not obtainable.

Since that date we find the following:—

Of the 39 patients who were known to be living, six are still alive, Nos. 35, 37, 64, 85, 89, 95.

Ten are dead, Nos. 58, 75, 84, 87, 88, 93, 97, 98, 99, 100, while there are two cases which were noted shortly after the paper was published as suffering from recurrence; these are Nos. 67 and 81. In the former there was local and general recurrence, and it is certain the patient did not live many weeks, while in the latter the patient had, when last heard

of, an enlarged liver, with nodules in the skin, so that we may fairly assume that both these died from spread of the disease, for which the eye was removed.

Patients still Alive.

Of the 103 cases, there are known to be but six patients living at the present time: they are the following:—

No. of case.	No. of years alive after operation.
35	17
37	16
64	$11\frac{10}{12}$
85	$9\frac{9}{12}$
89	$9\frac{6}{12}$
95	$8\frac{8}{12}$

6 cases.

The number of cases which cannot be traced is now 45 instead of 24, which was the number unaccounted for in 1891.

If we now examine the alleged cause of death in the ten cases in which a fatal result is recorded, together with the two in which we assume that death has taken place, we find as follows:—

No. of case.	No. of years alive after operation.	Alleged cause of death.
58	Unknown	Cause of death unknown.
75	Unknown	"Cancer of stomach."
84	About 4 years	General recurrence in liver, skin, &c.
87	$3\frac{7}{12}$	Recurrence in abdomen.
88	$2\frac{7}{12}$	"Died of cancer."
93	8	Cause of death unknown.
97	$6\frac{5}{12}$	Died of bronchitis. No recurrence.
98	3	"Died of cancer."
99	$4\frac{10}{12}$	Died of general recurrence.

No. of case.	No. of years alive after operation.	Alleged cause of death.
100	7	Died of cystitis, and hæmaturia, "probably due to malignant growth." No autopsy made.

Of the two we assume to be dead, the probable length of time that the patient lived, and the probable cause of death, was as follows:—

No of case.	No of years alive after operation,	Probable cause of death.
67	$11\frac{5}{12}$	Local and general recurrence.
81	About a year	General recurrence, skin, liver, &c.

If we add these 12 fatal cases to the list of 40 previously published, we find that 52 cases died out of 58 cases traced. Of these, 34 died of recurrent disease, giving a percentage of 58·62, this being a good deal larger than was found seven years ago, when it was 32·9 per cent.

These figures will again be referred to later on, when the more recent cases are taken.

Cases not previously Published.

We will now consider the cases which have occurred at this hospital since 1891, and we shall thus have 161 cases, extending over a period of 27 years.

Sex of Patients.

Of the 58 cases not previously reported, there were 24 males, 31 females, and three in which the sex was unknown. This gives a percentage of 41·38 males and 53·45 females, and 5·17 unknown.

The higher proportion of females is unusual. In the previous paper 57·28 per cent. were males, and 42·71 per cent.

were females, and in addition the authors go on to state of 259 cases recorded by Fuchs, there was a slight preponderance of males, while in only one paper by Martin containing a record of 43 cases, were there more women than men, 29 having occurred in women and 14 in men.

Age of Patients.

The following table shows in decades the ages of the patients :—

Years.	Males.	Females.	Total.
Between 20 and 30 there were	1	0	1
„ 30 „ 40 „	2	5	7
„ 40 „ 50 „	3	3	6
„ 50 „ 60 „	7	9	16
„ 60 „ 70 „	5	12	17
„ 70 „ 80 „	3	2	5
Age unknown	...	...	6
	21	31	58

The average age of the 52 known cases is 54·63. The youngest patient in this list was a man aged 28 years, No. 48, and the oldest a woman aged 74, No. 26.

Eye Affected.

Right eye	27
Left eye	28
Unknown	3
	58

Tension at the time of the operation, and the condition of the angle of the ant. chamber :—

Tension.	No. of cases.	Condition of angle.
T + 3	4	Closed in all.
T + 2	12	Closed in 11, narrowed in 1.
T + 1	6	Closed in 5, narrowed in 1.
T +	4	Closed in 1, narrowed in 2, not stated in 1.
T ? +	1	Open.
T n.	20	Open in 16. Closed in 1. Doubtful 1. Not stated 2.
T ? −	1	Not stated.
T −	2	Angle open in both.
T − 1	3	Angle open in all.
Not stated	2	
Globe solid with growth	2	
Globe bony and full of growth	1	
Total	58	

The following table is an analysis of the cases classed according to the tension of the eye at the time of operation, and the situation of the growth:—

Tension of eye.	Cases in which the ciliary body is involved.	Cases in which the choroid is affected without the ciliary body.	Total.
T + 3	2	2	4
T + 2	..	12	12
T + 1	3	3	6
T +	..	4	4
T + ?	..	1	1
T n.	10	10	20
T ? −	1	..	1
T −	1	1	2
T − 1	3	..	3
	20	33	
2 cases in which the globe was solid with growth, in both of which the ciliary body was affected 1 case in which the eye had bone in it 2 cases in which the tension was not stated			5
			58

We see from the above table that of the cases in which the tension was above normal, there were 22 in which the choroid only was affected, and only 5 in which the ciliary body was involved in the growth.

There was normal tension in an equal number of cases, while the tension was — in five cases where the growth involved the ciliary body, and in only one case in which the choroid only was affected.

The following is a summary of the table on tension :—

	Tumours of choroid.		Tumours of ciliary body.	
	Actual number.	Percentage.	Actual number.	Percentage.
Increased tension	22	66·66	5	25·00
Normal tension	10	30·30	10	50·00
Diminished tension ..	1	3·03	5	25·00

This again is confirmatory of a point brought out by the author in a paper before the Ophthalmological Society and published in the Transactions, vol. xvi, 1896, in which 100 cases of intraocular tumours were taken of all sorts, and it was found that, although the figures differed somewhat from the above, yet the general conclusions hold good, viz., that in growths which do not affect the ciliary body there is far more likelihood of the tension being increased than in cases in which the ciliary body is involved; in fact the tension is on a distinctly higher level in the former than it is in the latter.

The operations performed in these cases were as follows :—

In 56 cases ..... Enucleation of the eye only.
In 2 „ (50 and 57) Enucleation with subsequent extirpation of the contents of the orbit within a few days.

The following cases are known to have been alive for the number of years stated :—

No.				Years.
3	has lived	since	the excision	7
4	,,	,,	,, ...	7
5	,,	,,	,, ...	7
7	,,	,,	,, ...	$5\frac{4}{12}$
8	,,	,,	,, ...	$6\frac{3}{12}$
9	,,	,,	,, ...	$6\frac{1}{12}$
13	,,	,,	,, ...	$5\frac{5}{12}$
17	,,	,,	,, ...	$4\frac{1}{12}$
18	,,	,,	,, ...	3
19	,,	,,	,, ...	3
25	,,	,,	,, ...	$3\frac{1}{2}$
27	,,	,,	,, ...	$2\frac{8}{12}$
29	,,	,,	,, ...	$2\frac{8}{12}$
31	,,	,,	,, ...	$2\frac{9}{12}$
34	,,	,,	,, ...	$2\frac{5}{12}$
35	,,	,,	,, ...	$2\frac{5}{12}$
36	,,	,,	,, ...	$1\frac{2}{12}$
37	,,	,,	,, ...	2
38	,,	,,	,, ...	$2\frac{1}{12}$
41	,,	,,	,, ...	$1\frac{9}{12}$
42	,,	,,	,, ...	$1\frac{10}{12}$
44	,,	,,	,, ...	$1\frac{1}{2}$
46	,,	,,	,, ...	$1\frac{2}{12}$
47	,,	,,	,, ...	$1\frac{1}{12}$

There are 11 cases which have been operated upon within the last year, and these, as far as is known, are still alive; that is to say, there are 35 cases known to be alive out of the 58 in this series.

Of the 35 cases which are alive there are 11 which have remained free from recurrence for three years and upwards; the longest interval being in Cases 3, 4, and 5, in which a period of seven years has elapsed since the eye containing the tumour was removed.

We should, however, be careful to avoid drawing an arbitrary line such as a three years limit of recurrence, for cases do recur at a greater interval of time than this, such as is seen in Case 67 (first series), where the patient died of recurrence four years after the removal of the eye.

Causes of Death.

Six cases out of the total of 58 in this series are known to have died of recurrence; these are:—

Nos. 22, 23, 28, 30, 32, 45.

There are, however, 12 cases the fate of which is unknown. If we exclude these the percentage of deaths from recurrence amounts to 13·04.

The percentage found by Lawford and Collins was 32·9 out of the 79 cases which were traced, but since then the percentage has risen to 58·62.

Of the six cases in which there is no evidence that the patients died of recurrence there are two cases in which the cause of death is not known. These are Nos. 1 and 20.

The cause of death in the remaining four is given as follows:—

No. 14 died of "heart disease." Date unknown.
„ 21 „ "senile decay." 3 years after operation.
„ 26 „ "inflammation of kidneys." ? Metastatic growth. $1\frac{9}{12}$ years after operation.
„ 39 „ "strangulation of the bowel," nine months after operation.

The following 34 cases are known to be alive:—

Nos. 3, 4, 5, 7, 8, 9, 13, 17, 18, 19, 25, 27, 29, 31, 34, 35, 36, 37, 38, 41, 42*, 44, 46, 47, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58.

Of these Nos. 18 and 46 have Bright's disease, and they both have in addition albuminuric retinitis.

Situation of Recurrence.

Of the six cases in which there was known to be recurrence, No. 22 died in about $2\frac{1}{4}$ years of recurrence all over the body. No. 23 had local and general recurrence, and died five months after the eye was removed. No. 28 had recurrence "in the bone of her arm, which caused it to break,

* Patient has since died in S. George's Hospital of Sarcoma of the Liver, &c. The case is fully reported by Dr. H. D. Rolleston in The Lancet, May, 13, 1899.

necessitating amputation at the shoulder, after which the disease spread all over the body," and she died 14 months after the eye was removed. No. 30 died in S. Bartholomew's Hospital in February, 1897, 13 months after the removal of the eye, with recurrence in every part of the body, including a local recurrence in the orbit. She also had melanuria. No. 32 died in $1\frac{1}{3}$ years with recurrence in the lungs, abdomen, one of the bones of his leg (which nearly caused it to break), and all over the body. No. 45 died $1\frac{7}{12}$ years after the removal of the eye of "cancer of the liver." She also had a recurrence in the scalp, the nature of which was proved by microscopic observation after its removal during life. There was no local recurrence in the orbit.

Thus we see that two out of the six cases are known to have had local recurrence.

Six or exactly half (50 per cent.) of the cases which are known to be dead died of recurrence, and of these two had local and general recurrence, or 33·33 per cent., and in one of them (No. 23) the sclera had been perforated and the growth extended into the orbit before the eye was removed; while four had no local recurrence—that is to say, 66·33 per cent.

The proportion of local recurrence here is much larger than that found by Lawford and Collins, which was 8·86 per cent.

History of previous Injury.

In six cases a history of previous injury was obtained. Nos. 1, 22, 29, 50, 55, 58; while in No. 57 the affected eye is known to have been defective and divergent for many years through an unascertained cause.

Of the injured eyes No. 50 is the only one in which there is a definite history of a severe blow or wound. This was caused 30 years before removal by a blow from a skipping rope. The eye had remained quiet until a few months before removal, though quite blind. In addition there was a dense mass of bone in the choroid.

In No. 1 there is a history of a blow on the eye 30 years

ago with a gas pipe, followed by erysipelas, but from this the patient quite recovered, and the sight had failed for only three months.

No. 22 received a blow 12 years ago, but as the sight failed only one month before removal, it is not likely that any connection exists between the two events.

No. 29 had a slight injury four months before removal.

No. 55 was struck in the affected eye two years before the excision, immediately after which the sight failed. The patient, however, attended the hospital on account of pain which had been present for two weeks.

No. 58 had a slight injury to the eye 14 years before excision, but from this it quite recovered. The sight had, however, been failing for three years, and the eye had been quite blind for one year.

There is a history of injury in 10·34 per cent. of these cases, but in most of them the injury was probably a coincident and had nothing really to do with the malignant disease from which the patient suffered.

Considering, however, the fact that a lost eye containing bone is liable to become the seat of a malignant growth, and that frequently owing to the condition present the tumour is not able to be seen until it has existed for many months, one should not be too conservative in the treatment of blind eyes which after months or years of quiescence become painful. It will be found that in a large number of cases the secondary symptoms are due to malignant growths.

Family History.

With regard to the family history one cannot say very much, chiefly because of the somewhat unreliable statements which patients frequently make concerning their deceased relatives.

However, in a very large number of cases the history, so far as is obtainable, is absolutely negative. There are, however, four cases in which there is a history of malignant disease in the family, though in most of them there is no means of determining the nature of the "tumours" or

"cancers" from which the patients are supposed to have succumbed.

In No. 7 the father of the patient is said to have died of "cancer in the back."

In No. 18 the grandmother of the patient was "blind from tumour in the eye," and her (the patient's) mother had a "tumour of the neck."

In case 37 the patient's father died of "cancer of the bladder," and in No. 42 we are told that "there is a strong family history of cancer."

From such meagre information as this one cannot say that the tendency to sarcoma of the choroid is very marked in any of the families.

There is, however, one case which has been traced, and, as the family history is reliable, and as the disease affected many members of the family, it will be interesting to mention it here, especially as it completes a case which was included in the previous paper.

It is Case No. 99 in that series.

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of Patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
1. 3159	W. T.	27.6.91	Thomas Goulding	R.	56	Sight failing 3 months without pain.	Unknown
2. 3488	W. L.	28.8.91	(Middlesex Hospital case)	L.	..	..	..

On reference to this it will be seen that the patient is a daughter of Case No. 2, who had the left eye removed, together with some of the orbital contents for a melanotic sarcoma of the choroid which had perforated the sclerotic and was affecting the orbit. She became an in-patient at S. Bartholomew's Hospital suffering from abdominal recurrence, and she died about two years after the eye had been removed.

The best thanks of the author are due to Messrs. Nettleship, Tweedy, Lang, Morton, Rockliffe (Hull), Johnson Taylor (Norwich), Collier Green (Derby), and to the late Mr. Hodges, of Leicester, all of whom have provided cases which are included in this report, either from their private practice or from their practice at hospitals other than Moorfields to which they are attached, while most of those mentioned have spared no pains in finding out the subsequent history of their cases for the present paper. Mr. N. C. Ridley (Leicester) has also kindly given me some later notes of the cases which occurred in the practice of Mr. Hodges.

SARCOMA OF THE EYEBALL.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
·ad, but date and ause of death unnown.	Melanotic tumour involving base of iris, ciliary body, and choroid as far back as equator. Its cells are spindle-shaped, and the optic nerve is unaffected.	Tension normal.	..	Mother died of "internal cancer," aged 75 years. Affected eye injured with a gas pipe 30 years ago, followed by erysipelas. The sight quite recovered from this.
.. ..	Deeply pigmented growth reaching from base of iris to equator and inwards to middle of globe. It is a pigmented spindle cell sarcoma.			

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom or recurrence
3. 3493	R. M. G.	2.9.91	Daniel Thompson	R.	55	Sight failing 1 year. Blind 4—6 weeks. Pain 1 week.	..
4. 3509	A. S. M.	22.9.91	Mr. P. (Private patient)	R.	..	.. ..	..
5. 3525	Mr. Johnson Taylor	15.10.91	Mrs. G. (Private patient)	R.	60	..	..
6. 3534	Mr. Collier Green	26.10.91	Ellen T—— Case occurred at Derby General Infirmary.	L.	55	Sight failing for several weeks. Blind and very painful 1 week.	..
7. 3697	A. S. M.	3.5.92	Catherine Chapman	R.	30	Sight failing 1 month. No pain.	..
8. 3710	J. T.	17.5.92	Emily Chapman	L.	58	Sight failing 9 years. Blind and painful 1 year.	..
9. 3766	E. N.	30.7.92	John Dean	L.	60	Sight failing 1 year.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive and in good health 7 years after excision.	Pear-shaped melanotic sarcoma of choroid. O. N. is encroached upon, but not involved. Tumour is a mixed round and spindle cell growth not deeply pigmented.	T + 2, angle closed.	..	Eye injured with a piece of wood 3 weeks before excision. F. H. negative.
Alive and in good health 7 years after excision.	Mushroom-shaped growth with broad base nearly reaching O. N. behind and O. S. in front. Slightly pigmented small spindle cell sarcoma. Sclera somewhat involved.			
Alive and in good health 7 years after excision.	Two tumours in ciliary region on opposite sides, each size of a pea. Small spindle cell sarcomata containing pigment.	T. normal.	..	No known history of tumour in the family.
.. ..	Mushroom-shaped growth of choroid extending from close above O. D. to equator.	T. + 2, angle closed.		
Alive and well $5\frac{4}{12}$ years after excision.	Tumour reaches from ciliary body to within 4 mm. of O. D. O. N. healthy. Slightly pigmented spindle cell sarcoma.	T. normal, angle open.	..	Father died of "cancer in the back." No history of injury or previous eye disease.
Alive and well $6\frac{3}{12}$ years after excision.	Melanotic mixed cell sarcoma arising from choroid around O. D.	T. normal, angle open.	..	F. H. negative. No history of injury or previous eye disease.
Alive and well $6\frac{1}{12}$ years after excision.	Unpigmented sarcoma growing from choroid on inner side, reaches from O. D. nearly to O. S. Tumour involves ciliary artery and extends into it.	T. normal, angle open.	..	Slight injury 2 years ago.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
10 3812	A. S. M.	17.9.92	Sarah Jackson	L.	56	Sight failing 9 months. Blind 4 months. Painful 6 weeks.	..
11 3854	J. B. L.	16.10.92	George Jackson	R.	37	Eye blind 15 months. Painful 1 week.	..
12 3933	J. C.	28.2.93	Eliza Clements	L.	46	Sight failing without pain for 2 months.	..
13 3962	Mr. Johnson Taylor	9.3.93	Sarah R. (Private patient)	R.	53	..	..
14 4007	W. L.	16.5.93	Charles King	L.	62	Sight failing 1 year. Blind 5½ months. Pain 3 weeks.	..
15 4029	E. N.	17.6.93	Miss M. J. B.	R.	63	Flickering before eyes 1 month.	..
16 4141	J. C.	27.10.93	Sarah Haddon	L.	52	Sight failing 6 months without pain.	..
17 4354	W. T.	9.7.94	Helen Chalcroft	R.	63	Sight failing 1 year with occasional attacks of pain.	..
18 4428	A. S. M.	2.10.94	Annie Kiff	R.	30	Sight failing for 20 months. No pain.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence	Family history, remarks, &c.
Not able to be traced.	Slightly pigmented sarcoma growing from the outer half of the choroid.	T. + 2, angle closed.	..	F. H. negative.
..	Large pigmented growth on outer side of globe reaching nearly forwards to lens. Retina detached elsewhere.	T. +, angle narrowed.	..	F. H. negative. Not able to be traced.
..	Melanotic spindle cell sarcoma reaching from ciliary body to equator.	T. normal, angle open.	..	F. H. negative. No history of injury or of previous disease. Not able to be traced.
Alive and well $5\frac{5}{12}$ years after excision.	Melanotic sarcoma of posterior part of choroid close to O. D. and Y. S. Small spindle cell growth	T. normal, angle open.	..	F. H. unknown.
"Died some time ago of heart disease."	Small spindle cell melanotic sarcoma of choroid reaching from O. D. to equator. O. N. not affected.	T. + 2, angle narrowed	..	F. H. negative. No history of injury or of previous disease.
..	Melanotic sarcoma reaching from ciliary body to O. N. Small round and spindle cell growth.	T. ? −	..	F. H. unknown. Not able to be traced.
..	Slightly pigmented small round and spindle cell sarcoma growing from choroid.	T. +, angle narrowed	..	F. H. negative. No previous injury or disease. Not able to be traced.
Alive and well $4\frac{1}{12}$ years after excision.	Small round and spindle cell melanotic sarcoma of choroid. O. N. healthy.	T. normal, angle open.	..	F. H. negative. No previous injury or disease.
Alive 3 years after excision, but has albuminuric retinitis in the other eye.	Mixed round and spindle cell pigmented sarcoma of choroid reaching from near ciliary body to O. D.	T. full, angle greatly narrowed.	..	Grandmother blind from tumour of the eye. Mother has tumour of neck.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
19 4412	R. M. G.	17.10.94	Alfred Gibbons	L.	43	Sight failing 4 years. No pain.	..
20 4477	A. Q. S.	3.12.94	Mary Dorrington	R.	61	Sight failing 2 years. Pain 1 month.	..
21 4481	Mr. Hodges, Leicester Infirmary.	6.12.94	John Bates	R.	73	Eye blind for 16 years. Pain 4 months.	..
22 4483	W. T.	13.12.94	Edward Rittman	L.	45	Sight failing 1 month.	About $2\frac{1}{4}$ years.
23 4486	A. S. M.	14.12.94	Annie Carter	R.	40	Sight failing 3 years. Pain 3 weeks.	A few weeks.
24 4 O.P.	A. S. M. G. N. Hosp.	12.2.95	..	..	..	..	..
25 4561	A. Q. S.	21.3.95	Harriet Godfrey	R.	61	No symptoms were noticed by patient.	..
26 4684	A S. M.	30.8.95	Theriza Horn	L.	74	Eye affected "some time." Occasional pain.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive 3 years after excision, but in bad health. Suggestive of abdominal recurrence. No local recurrence.	Unpigmented sarcoma close to O. N. Sclera much thinned and almost perforated. It is a small round cell growth.	T. normal, angle open.	..	F. H. negative. No injury or previous disease.
1 year	Melanotic sarcoma, nearly filling globe.	T. +2 angle closed.	..	F. H. negative. Cause of death unknown.
2 years. Died of "senile decay." No recurrence.	Globe full of a dense round cell melanotic sarcoma, which has perforated sclerotic near equator. Large extra-ocular mass nearly as large as the eye.	Globe solid.	..	F. H. unknown.
About 2¼ years ..	Rather large mottled sarcoma of choroid. It is a round and spindle cell growth.	T. normal, angle open.	Liver, lungs, and probably other viscera.	F. H. negative. Blow 12 years ago; probably not a severe injury.
4 months	Large flattened melanotic sarcoma of choroid which has perforated sclera near O. N. Large mass in orbit.	T. +3, angle closed.	"Cancer developed all over her including the orbit."	Father died of cancer. No injury or previous eye disease.
..	Round and spindle cell melanotic sarcoma, extending from O. D. to about equator.	Angle open.		
Alive and well 3½ years after excision.	Sarcoma the size of a pea in ciliary region with a large cyst developed in it.	T. normal, angle open.	..	F. H. negative.
9 months	Melanotic sarcoma in ciliary region pushing the iris forwards.	T. normal, A. C. shallow.	..	F. H. negative. Patient died in May, 1897, from "inflammation of the kidneys."

Sarcoma of the Eyeball.

No. of case and No. in Hosp Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affec-d.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence
27 590.P.	Mr. Rockliffe (Private case).	22.10.95	Mr. S. P. H.	L.	42	Sight failing for a "few weeks."	..
28 4748	W. L.	18.11.95	Emma Topsfield	L.	64	Eye weak for 4 to 5 years. Occasional pain.	Unknown
29 4765	R. M. G.	7.12.95	Emily Gooding	L.	60	Sight failing 4 to 5 months.	..
30 4779	E. T. C.	26.12.95	Eliza Thacker	R.	70	Sight failing 2 months. Blind and painful 3 weeks.	6 weeks
31 4782	A. S. M.	31.12.95	Mary Nash	R.	33	Sight failing 6 months.	..
32 4795	J. B. L.	8.1.96	Charles Piper	R.	71	Sight failing for many months. Blind 1 year. Pain 6 weeks.	Unknown
33 750.P.	J. T. Case at U. C. H.	28.2.96	John Reed	..	..	..	..
34 4844	W. T.	9.3.96	Mary Radley	L.	55	Sight failing 3—4 months.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive and in good health $2\frac{8}{12}$ years after excision.	Unpigmented sarcoma of choroid composed of mixed round and spindle cells, and extending from O. N. to equator.	T. normal, angle open		
4 months	Melanotic sarcoma reaching from ciliary body to within 3 mm. of O. D. Mixed round and spindle cell growth.	T. −, angle open.	General recurrence all over body.	Before death the arm fractured and was amputated "for cancer."
Alive and well $2\frac{8}{12}$ years after excision.	Unpigmented small round cell sarcoma growing from ciliary body and reaching to equator.	T. normal, angle open.	..	Slight injury 4 months before excision.
3 months	Melanotic sarcoma extending from iris to within 15 mm. of O. N. O. N. healthy.	T + 3, angle closed.	Local and general recurrence in every part of body.	Patient had melanuria, and died in St. Bartholomew's Hospital. F. H. negative.
Alive and well $2\frac{9}{12}$ years after excision.	Melanotic sarcoma reaching from ciliary body to posterior pole of eye.	T. −1, angle open.	..	No injury. F. H. negative.
6 months	Melanotic sarcoma reaches root of iris and half fills globe; this has given way on one side, and there is a large extra-ocular mass.	T. + 3, angle closed.	General recurrence May, 1897.	Patient had recurrence in one of the bones of the leg which nearly caused it to break, as well as all over the body.
..	Unpigmented sarcoma occupying region of O. N.	Angle closed.		
Alive and well $2\frac{5}{12}$ years after excision.	Melanotic sarcoma in ciliary region about the size of a split pea. Small round cell growth.	T. + 1. Angle open on one side, closed on the other.	..	F. H. negative. No previous injury.

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
35 4851	J. T.	17.3.96	Jane Dalgleish	R.	37	Sight failing several years. Blind 2 months.	..
36 4928	E. N.	14.6.96	James Campbell	R.	55	Sight failing 1 year. Blind 3 weeks.	..
37 4991	A. S. M.	18.8.96	Robert Woodcraft	R.	61	Sight failing 5—6 weeks.	..
38 4969	R. M. G.	25.7.96	Edmund Howard	L.	63	Sight failing 3 years. Blind 5 months. Pain 1 month.	..
39 4993	E. T. C.	20.8.96	James Wastell	R.	69	Sight affected for 8 months.	..
40 137 O.P.	Mr. Rockliffe. Case from Hull.	31.10.96	Sarah B.	L.	50	Acute glaucoma for 3—4 weeks.	..
41 5048	E. T. C.	12.11.96	Mary Jenkins	R.	49	Sight failing for 8 months. No pain.	..
42 137 O. P.	A. S. M.	15.11.96	Mr. B.	L.	About 50	Sight failing for 7 weeks.	..
43 141 O. P.	Mr. Rockliffe. Case from Hull.	21.11.96	.. ..	..	..	.. ..	..
44* 5113	W. L.	18.2.97	William Dumsday	R.	57	Sight failing 3 months.	..

* See footnote, page 59.

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive and well $2\frac{5}{12}$ years after excision.	Melanotic sarcoma reaching from near O. N. to equator.	T. normal, angle open.	..	F. H. negative. Patient not very strong.
Alive and well $1\frac{2}{12}$ years after excision.	Small round cell melanotic sarcoma growing from region of equator.	T. +.	..	F. H. negative. No history of injury.
Alive and well 2 years after excision.	Slightly pigmented small round cell sarcoma reaching from ciliary body to equator.	T. −, angle open.	..	Father died of cancer of the bladder. No history of injury or previous eye trouble.
In good health $2\frac{1}{12}$ years after excision.	Unpigmented spindle cell sarcoma which is invading the sclerotic at one place.	T. +2.	..	F. H. negative. No previous eye affection.
9 months	Large melanotic sarcoma reaching from ciliary body to O. D. Small round and spindle cell growth.	T. normal, angle open.	..	F. H. negative. No injury. Patient died of "strangulation of the bowel," May, 1897.
.. ..	Mushroom-shaped melanotic sarcoma growing from choroid.	T. +2, angle closed.	..	Unable to be traced.
Alive and well $1\frac{9}{12}$ years after excision.	Slightly pigmented sarcoma; it is thin and flat.	T. normal, angle open.	..	F. H. negative. No injury or previous eye disease.
Alive and well $1\frac{10}{12}$ years after excision.	Melanotic sarcoma extending from ciliary body to within 5 mm. of O. D.	T. −1, angle open.	..	Strong family history of cancer.
.. ..	Melanotic sarcoma of ciliary body and of choroid.	Angle closed.	..	Unable to be traced.
Alive and well $1\frac{1}{2}$ years after excision.	Melanotic sarcoma extending from edge of O. N. to equator.	T. normal, angle open.	..	F. H. negative. No injury or previous disease.

Sarcoma of the Eyeball.

No. of case and No in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence
45 5127	W. L.	1.3.97	Caroline Slade	L.	67	Sight failing for 18 months. Much pain.	11 months
46 5214	E. N.	19.6.97	Jane Brown	R.	65	Sight failing 1 year. Pain recently.	..
47 5231	E. N.	10.7.97	James Cooper	L.	56	Sight failing 9 months. No pain.	..
48 5232	E. T. C.	12.7.97	Frank Phillips	L.	28	Sight failing 4 months. Painful 4 days.	..
49 5309	E. N.	16.10.97	Rosana Bentley	L.	60	Detachment seen 11 months. Pain 1 week.	..
50 5366	W. T.	6.1.98	Miriam Woolley	R.	50	Eye blind and shrunken 30 years. Painful 3 months.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Died $1\frac{7}{12}$ years after excision.	Large round and spindle cell melanotic sarcoma filling two-thirds of globe. Ciliary body not affected.	T. +1, angle closed.	Liver and scalp.	F. H. negative. No injury. General recurrence.
Alive and well $1\frac{2}{12}$ years after excision.	Large melanotic sarcoma composed of small round cells extending from O. N. on one side to within 3 mm. of the ciliary body on the other.	T. +2, angle closed.	..	F. H. negative. No injury. She has albuminuric retinitis.
Alive and well $1\frac{1}{12}$ years after excision.	Slightly pigmented small round cell sarcoma. Ciliary body not involved.	T. −, angle open.	..	F. H. negative. No injury or previous eye disease.
..	Large melanotic sarcoma with round and spindle cells; it is growing from the choroid and just reaches the ciliary body.	T. +2, angle closed.	..	F. H. negative. Patient not able to be traced.
In good health 10 months after excision.	Two nodules of growth outside sclerotic near superior oblique insertion. Mottled growth on disc connected with small growth outside; also flat mass extending from this, 0·5 mm. thick occupying posterior third of eyeball. Slightly pigmented round cell sarcoma, and affects head of O. N.	T. +2, angle closed.	..	F. H. negative. No injury. The growth is remarkably diffuse, and gave rise to the appearance of retino-choroditis ophthalmoscopically.
Alive 1 year after excision.	Eye solid, with a large mass of bone inside it. Surrounding this and extending outside as two large masses is a coal-black sarcoma. O. N. surrounded by growth. Lens calcareous.	..	..	F. H. negative. Eye injured with skipping-rope, 30 years before removal. It had been blind since. Contents of orbit removed.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
51 5380	R. M. G.	19.1.98	Mary Jerrell	L.	67	Two or three attacks of acute glaucoma during 8 months.	..
52 5424	J. B. L.	2.4.98	Thomas McCombe	L.	73	Sight failing 7 years. Blind 5 years. Old iritis.	..
53 5481	A. Q. S.	28.6.98	Charles Pain	L.	34	Sight noticed to be failing 3 weeks.	..
54 5488	J. T.	5.7.98	Louisa Cheshire	L.	57	Sight failing 10 months. Blind 7 months.	..
55 5491	W. T.	7.7.98	Susan Sheppard	R.	39	Sight failed 2 years ago after a blow. Pain 2 weeks.	..
56 5501	W. L.	21.7.98	Maria Kemp	L.	61	Sight failing 5 years. Pain 7 months.	..
57 5511	T. W. H. S.	6.8.98	William Dewsberry	L.	55	Eye defective and divergent for 20 years or more. Occasional pain for 4 years.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive $\frac{11}{12}$ after excision.	Large mottled sarcoma with round and spindle cells. It extends from a point 7 mm. from O. D. on one side to within 3 mm. of O. S. on the other side.	T. +2. angle closed.	..	F. H. negative. No injury.
Quite well 4 months after excision.	Large melanotic sarcoma with round and spindle cells growing by a pedicle from O. D. and reaching lens. Ciliary body not involved.	T. +2, angle closed.	..	F. H. negative.
Alive $\frac{6}{12}$ after excision.	Unpigmented mixed round and spindle cell sarcoma growing from equator at upper part.	T. normal, angle open.	..	F. H. negative.
Alive $\frac{5}{12}$ after excision.	Melanotic sarcoma, filling two-thirds of the subretinal space.	T. +1, angle closed.	..	F. H. negative. No injury or previous eye disease.
Alive $\frac{5}{12}$ after excision	Rather large sarcoma reaching from O. D. nearly to equator. Free from pigment. Small round cell growth. Broken down with hæmorrhage.	T. +3. No A. C. Angle closed.	..	F. H. negative. Sight failed after a blow 2 years before excision.
Alive $\frac{5}{12}$ after excision.	Small melanotic sarcoma near equator. Centre broken down with hæmorrhage. Much degeneration.	T. +2, angle closed.	..	F. H. negative. Detachment of retina was seen 5 years before excision.
Alive $\frac{4}{12}$ after excision.	Eye completely solid and filled with a dense black sarcoma. Centre is soft and broken down. Sclerotic has given way behind and growth projects into orbit.	..	..	F. H. negative. Contents of orbit removed.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
58 5542	W. L.	8.9.98	Harry Goodchild	R.	50	Sight failing 3 years. Blind 1 year. Slight pain for 3 weeks.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive $\frac{3}{12}$ after excision.	Small mushroom-shaped melanotic sarcoma growing from posterior part of choroid. Retina completely detached.	T. full, angle closed.	..	F. H. negative. Slight injury 14 years before excision, but from this it quite recovered.